THE
BRITISH JOURNAL OF SURGERY

VOL. XXXVII JULY, 1949 No. 145

THE TREATMENT OF MALIGNANT OBSTRUCTION OF THE CARDIA

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THERE have been 310 patients with malignant obstruction of the gastro-œsophageal junction investigated and treated in the thoracic surgical department at Leeds up to June, 1948. At the same time there have been 96 examples of peptic ulcer of the lower œsophagus, 90 of which caused dysphagia; 56 obstructions due to cardiospasm, 1 from a simple peptic ulcer of the stomach, and 1 from a simple tumour which turned out to be a huge leiomyoma. In this series, squamous carcinoma of the œsophagus was about twice as common as peptic ulcer in the same site. Although there has been notable improvement in the diagnosis and treatment of these patients, it cannot be denied that the main bastion of the problem stands unassailed, for out of the total of 310 only 121 were fit for any surgical assistance. The results of treatment, however, cannot be shown by survival rates alone. There are no numerals for human suffering and the extent of its relief cannot be reduced to a set of figures. The aim of treatment has usually been to overcome obstruction so that the patient could continue to swallow his food and saliva until he died. Stenosis of the lower end of the œsophagus causes misery not only from starvation but from the collection and regurgitation of food and saliva. When the growth starts in the stomach there may be some loss of energy, anæmia, and loss of weight for months before dysphagia appears, but this is not always so. Many of the patients declared that they had had a mild obstruction weeks or months before the onset of persistent symptoms but that this had cleared and had been ignored both by themselves and their doctor. The importance of investigation by all the methods available at this early stage must be emphasized. Too often dysphagia is ignored when it passes off spontaneously, or reassurance is based on a too cursory examination.

PALLIATIVE TREATMENT

Many patients were almost moribund by the time they were seen and the administration of morphine was almost all that could be done for them. The aspiration of the œsophageal contents through a test-meal tube was helpful, especially in the evening, so that the patient was not kept awake by frequent regurgitation.

Gastrostomy and jejunostomy are condemned. The operation carries a considerable mortality, it adds to the discomfort, there is no evidence that it prolongs life, and it does not relieve the patient's distress. The only possible advantage is that it relieves starvation, but this can be done so much better by intravenous feeding and œsophagoscopic dilatation of the stricture. The most satisfactory palliative measure is the admittedly rather crude one of passing bougies under direct vision until the lumen is wide enough to take a Souttar's tube (*Fig.* 1). Although this causes trauma to the growth and sometimes moderate oozing of blood, it can be done with safety if a stylet with a gum-elastic end is first passed into the stomach and then metal dilators passed over it. The Souttar's tube also is passed over the stylet so that there is no danger of making a false passage. A fluid and soft, solid diet can be taken through this. It not only relieves thirst and starvation, but it is a great mental relief to the patient and gives him the continued joy of taking his food by mouth. In this way a good life has been prolonged and enjoyed for as long as eighteen months. After a variable time the tube works its way through the stricture and is passed by

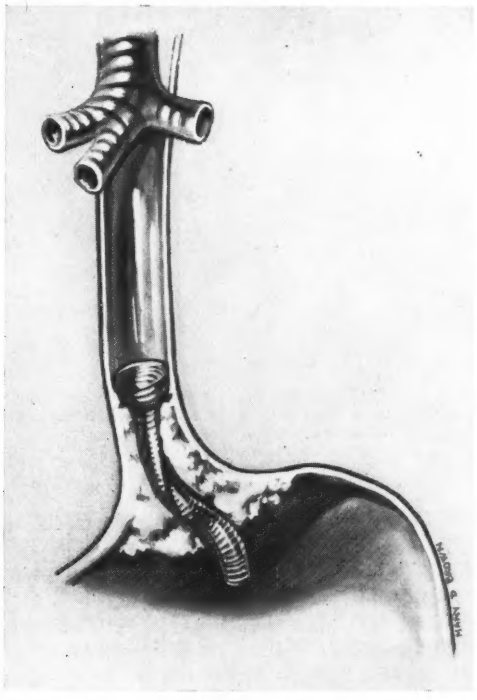
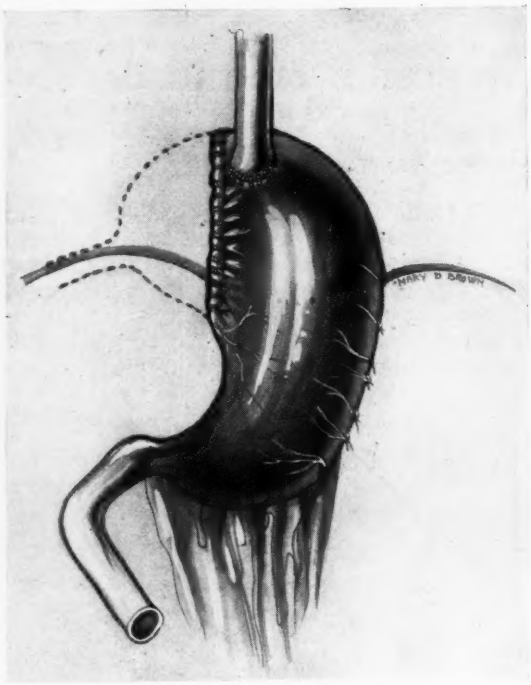


FIG. 1.—Souttar's tube through malignant stricture of lower œsophagus.

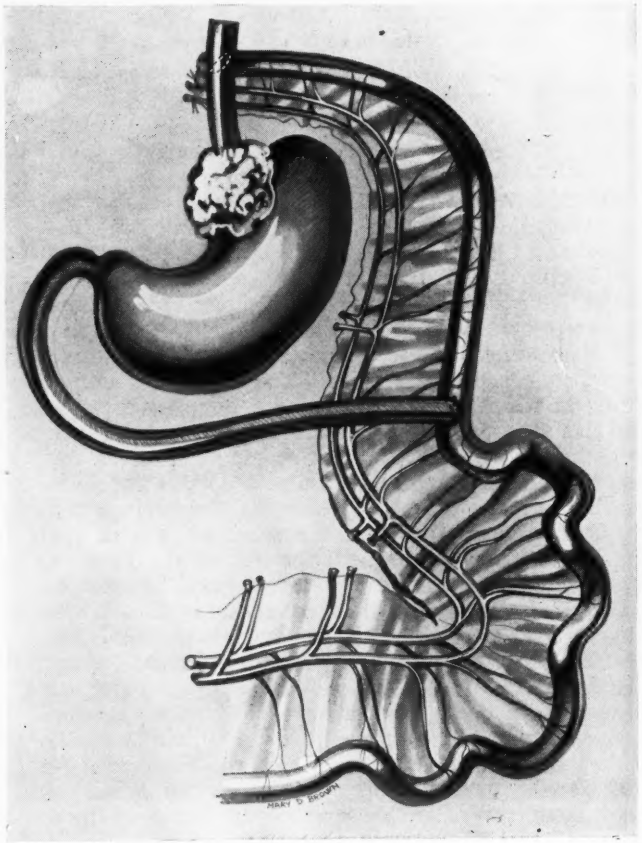
the bowel. A fresh tube may then have to be introduced. Trouble may occur when the growth extends upwards or downwards over the end of the tube, when it may have to be removed and a larger tube inserted. Sometimes ill-chewed food may become impacted in the lumen of the tube and cause obstruction. It is always worth trying to displace this with an effervescent drink before the necessity for œsophagoscopy is considered. Even with these troubles and the ultimate fatal outcome the picture is a much brighter one than that of an abdominal operation, gastrostomy feeding, and the accumulation of saliva in the œsophagus, with frequent regurgitation.

Radiotherapy as a palliative measure either by the usual exposures or by direct application has not yet done more than support the hope that it may be improved. The untoward effects on the liver involved in the treatment of growths in this region make it unsatisfactory; but its full possibilities, especially in association with surgery, have yet to be explored.

Surgery itself does play a part in the palliation of this condition. It may sometimes be advisable to do a set palliative operation, but more often an operation is undertaken with a view to extirpation of the growth, and the impossibility of this is only found out when it is exposed. In the latter circumstances something to relieve œsophageal obstruction should be done, for it is the patient's reasonable expectation that after a big operation for dysphagia he should at least be able to swallow his food even if he cannot be cured of his disease. If the primary growth can be removed this should be done, followed by anastomosis in the mediastinum of the distal half of the mobilized stomach to the cut end of the œsophagus (*Fig.* 2 A). If the growth is not only inoperable but also irremovable, it should be side-tracked by anastomosing the œsophagus above it to a loop of jejunum fashioned according to the method of Roux (*Fig.* 2 B).



A



B

FIG. 2.—A, Palliative œsophago-gastrostomy after removal of primary growth; B, Palliative œsophago-jejunostomy for irremovable growth at the cardia.

RADICAL TREATMENT

Growths of the cardia have been approached from the abdomen and from the chest. The direction of attack is liable to be decided according to the surgeon's familiarity with a particular incision, and his operation is then limited by his reach. This in turn is governed by his dexterity and his persistence. The results of the surgical treatment of carcinoma anywhere in the stomach by the most skilled surgeons are very bad, and one of the factors in this has been the limitations of the abdominal approach. The time is opportune, therefore, to review gastric growths and those of the lower end of the œsophagus to see how they spread, and to practise a method of excision which is as sound in theory as the abdomino-perineal excision for rectal carcinoma. If these requirements are accepted there can only be one sound surgical approach, the

thoraco-abdominal. Although these considerations are of carcinoma involving the gastro-œsophageal junction, they are relevant to growths occurring anywhere in the stomach, for the lesser curvature is so short that it cannot be thought of in sections when it is involved by malignant disease any more

FIG. 3.—Infiltrating squamous carcinoma of lower third of œsophagus invading stomach wall, diaphragm, and mediastinal cellular tissue.

than the segments of the breast can be taken separately in carcinoma of this organ.

The Spread of Carcinoma of the Lower Third of the Œsophagus.—Growth starting in the squamous epithelium of the lower end of the œsophagus spreads through the muscle wall and into the tissues of the mediastinum, where it becomes fixed to the aorta, either or both pleuræ, adjacent lung, diaphragm, and pericardium. Involvement of the aorta has been considered an absolute obstruction to removal, but parts of all the other structures may have to be resected. Examples of growths perforating into the lung and causing fatal gangrene have been observed, but short of this there may be minimal attachments where removal of a segment of lung may be required to leave a safe margin beyond the advancing edge of the tumour. The mediastinal tissue should be taken with the œsophagus as a routine, and there should be no hesitation in including adequate areas of mediastinal pleura. Spread also occurs upwards and downwards in the wall of the œsophagus. The junction of the œsophagus and stomach is no barrier to the spread of carcinoma, and when ulceration is present on both sides of the cardia it may need histological examination to decide in which organ it started (*Fig.* 3). The upward spread in the œsophageal wall is particularly interesting and important. It may be a uniform, superficial spread extending up to the level of the

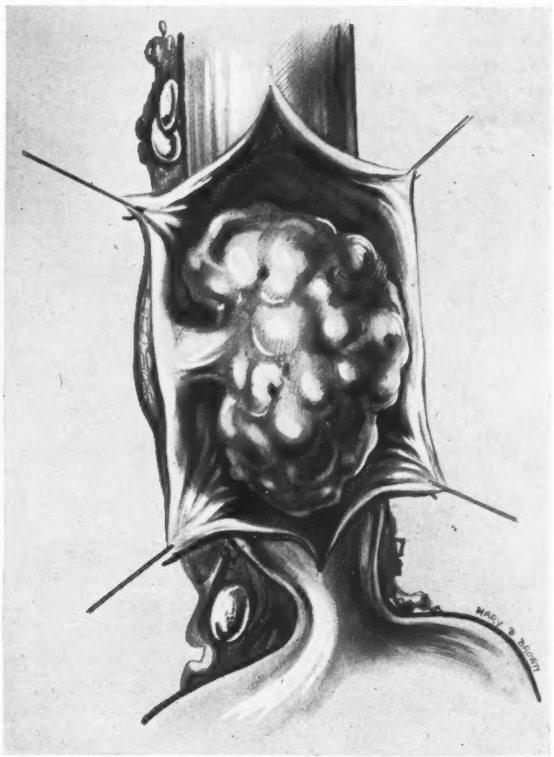


FIG. 4.—Pedunculated squamous carcinoma of œsophagus forming huge mass in the lumen of the lower third of the œsophagus attached by only a small pedicle.

left main bronchus or the arch of the aorta, with less extensive spread in the deeper muscle layers, or there may be islands of growth at some distance from the main mass and surrounded by apparently normal epithelium. These islands may be numerous and would certainly not be detected either radiologically or by palpation at operation. They stress the necessity for the most careful œsophagoscopic examination before operation and for the resection at operation of an ample length of œsophagus above the palpable growth. There is some doubt if these islands have in fact spread from the main growth. It seems sometimes that the process is a widespread patchy malignant degeneration of the epithelial lining of this part of the œsophagus. Whatever may be the explanation of these findings the practical application is that it may be necessary to resect the œsophagus as high up as the arch of the aorta for a growth which is palpable only at the lower end. Occasionally the main direction of growth is into the lumen itself. It then forms a malignant polyp which is made up of compact lobules and tends to become sausage-shaped (*Fig.* 4). Although it may show radiologically as an extensive mass and appear

most formidable through the œsophagoscope, there may be only little infiltration of the œsophageal wall and it may be easily removable. These soft masses do not cause very marked dysphagia, so that by the time they are treated the lymphatic involvement is often widespread. Polypoidal growths tend to become necrotic and nearly the whole of the mass within the lumen may slough away. If they occur close to the cardia they may prolapse into the stomach and then grow as a cauliflower-like mass in the fundus.

The spread to lymphatic glands is primarily into those of the posterior mediastinum. These occur on all aspects of the œsophagus and extend into the lateral pulmonary ligaments. The main flow is downwards into the glands around the cardia and along the left gastric group to those around the cœliac axis. At the cardia cells may be diverted into the suprapancreatic glands, and malignant invasion of glands along the pancreas have been seen even when the local mediastinal glands have not been involved (*Case* 9, *Figs.* 30, 31). Although the main direction is downwards there is also a lymphatic spread upwards to the posterior mediastinal, tracheo-bronchial, and cervical glands. These last may be detected on clinical examination and so forestall surgery, but the others may be found only when the chest is opened. They may, of course, easily be missed even by direct examination of the mediastinum.

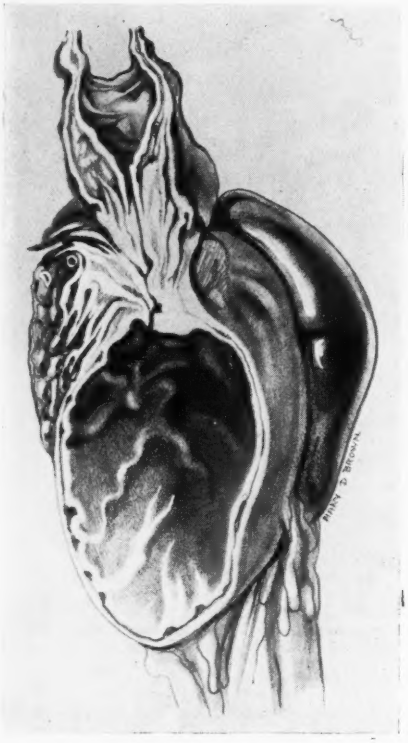


FIG. 5.—Carcinoma of stomach involving pancreas by direct spread.

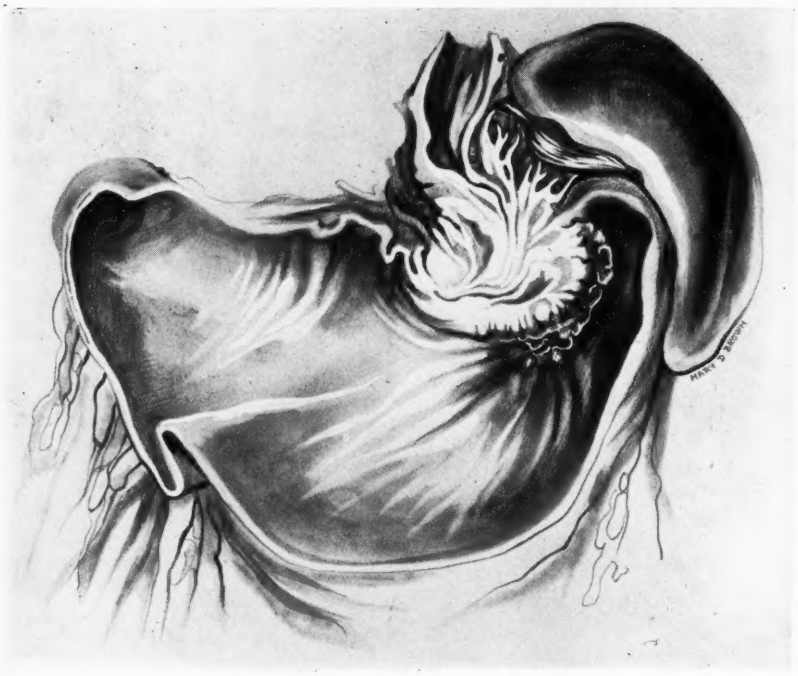


FIG. 6.—Carcinoma of stomach invading diaphragm.

The Spread of Carcinoma of the Cardiac End of the Stomach.—Carcinoma of the stomach spreads within the wall of the stomach itself and is detectable histologically at varying distances from the visible and palpable margin of the growth. The extension may be in the tissue spaces or in the lymphatic plexuses, particularly in the submucosa, but it has also been observed in the perineural spaces. When it reaches the cardia it spreads up into the œsophagus. As the advancing edge of the growth is usually in the submucosa its spread around the lower end of the œsophagus tends at one stage to produce a smooth funnel-shaped obstruction here which is easily mistaken for cardiospasm. Extension outwards involves the muscle layers and the peritoneum of the stomach itself, whence it can spread diffusely in the peritoneal cavity or become adherent to neighbouring structures. In the latter event it may involve the pancreas, diaphragm, spleen, left suprarenal capsule, or the left lobe of the liver (*Figs.* 5–9). Direct invasion of any of these organs is no obstruction to successful removal, and examples of each have been encountered in the present series. As with carcinoma of any hollow viscus, the main mass of growth may be in the lumen itself, so that the size of the tumour on radiological examination is no guide to its operability.

The lymphatics of the stomach were investigated by Jamieson and Dobson (1907), but the limitations of the abdominal or the thoracic approaches to gastric carcinoma led surgeons to ignore this classic contribution to lymphatic anatomy (*Figs.* 10, 11). The lymphatic vessels of the lesser curvature drain mostly upwards towards the cardia, where they pass back into the retroperitoneal tissue and follow the course of the gastric vessels to the cœliac axis. Some vessels from the pyloric antrum pass into the free edge of the lesser omentum and some to the adjacent

suprapancreatic region. Vessels from the fundus drain partly towards the cardia, partly over the top to the suprapancreatic region, and partly through the gastrosplenic omentum and lieno-renal ligament

Gland stations are found all along the curvatures of the stomach, but are described in groups: those along the lesser curvature are known as the lower group of the superior gastric glands; those around

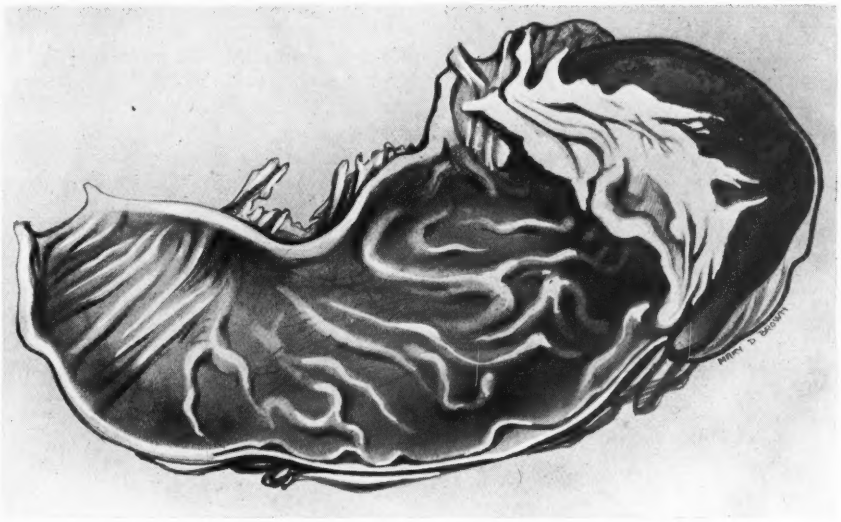


FIG. 7.—Carcinoma of stomach invading spleen.

to the retroperitoneal tissue. From the region of the cardia there is a flow upwards along the œsophagus to the posterior mediastinum. The vessels from the lower part of the greater curvature drain towards the pylorus, following the course of the right gastro-epiploic vessels.

the cardia which lie like a chain of beads are the paracardial group; those near the hilum of the spleen form part of the pancreatico-lienal group; whilst those on the greater curvature are the inferior gastric glands. The next lot of gland stations are found in the retroperitoneal tissues and posterior mediastinum, the former occurring mainly along the upper border of the pancreas and in the subpyloric region, the latter lying around the œsophagus

FIG. 8.—Carcinoma of stomach invading left suprarenal capsule.

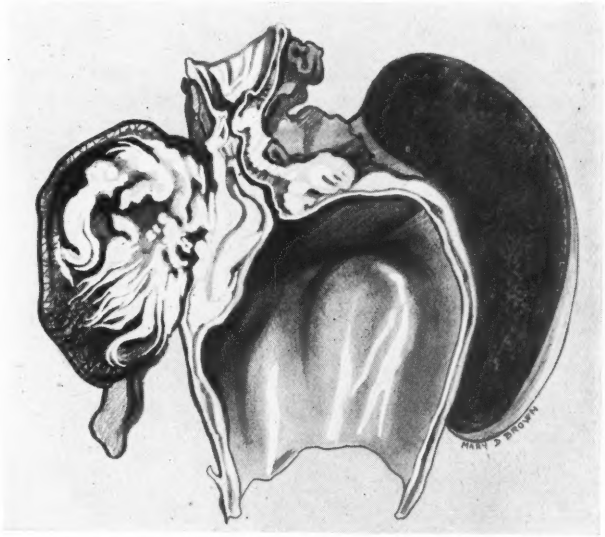


FIG. 9.—Carcinoma of stomach invading left lobe of liver.

immediately above the diaphragm like the paracardial group below and extending upwards from here to the root of the neck. It is well known that malignant cells may by-pass the nearest gland stations, and in one patient who came to post-mortem without operation a gastric carcinoma had involved

a lymphatic gland on the right side of the œsophagus where this is crossed by the arch of the azygos vein and there was no evidence of any other involvement of the posterior mediastinum.

Requirements of a Radical Operation for Carcinoma of the Upper Half of the Stomach.—The theoretical points discussed above have been repeatedly tested during the course of operative

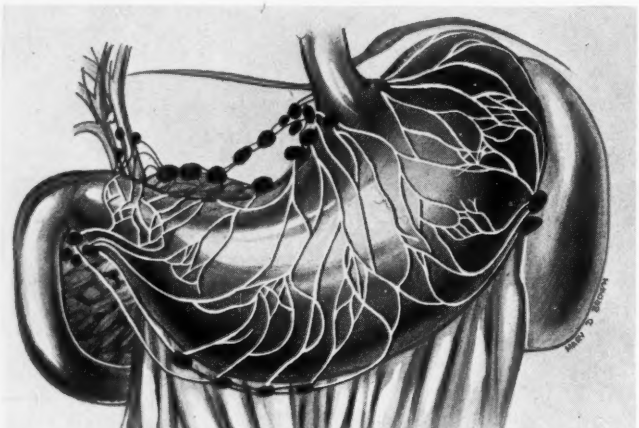


FIG. 10.—Lymphatics of the stomach as seen from front. (*After Jamieson and Dobson.*)

dissections and on post-mortem material. The radical operations to be suggested are the practical expression of their proven validity. In the earlier operations it seemed to be enough to have resected the growth and kept the patient alive, and where cure seems impossible this may be fully justified. The return of patients with recurrence a few months after removal of an apparently operable growth soon undermines any satisfaction that may be felt from a low initial mortality, and the more recent patients have been subjected to a radical procedure in the belief, so ably expressed by Sir James Walton, that " too much attention can be paid to the prolongation of life and too little to its quality ". A block dissection has been performed to include the whole of the stomach and lower end of œsophagus, the great omentum, the lesser omentum flush with the liver and medially up to the hepatic artery, the spleen, the pancreas with retroperitoneal tissue and glands as far as the neck, the left gastric vessels and surrounding tissue as far back as the cœliac axis, a cuff of diaphragm around the cardia, and the posterior mediastinal cellular tissue as high as the inferior pulmonary vein. Although this is a big mass of tissue the dissection can be done in tissue planes with very little bleeding and with practically no contact with the growth. The duodenum is closed and the œsophagus anastomosed to the jejunum.

Requirements of a Radical Operation for Carcinoma of the Lower End of the Œsophagus.—The block dissection for carcinoma of the œsophagus is similar to that described for the stomach, but some modifications are used. The length of œsophagus removed is greater for reasons already explained, but only the fundus, cardia, and lesser curvature of the stomach are resected. The rest of the stomach is closed and anastomosed to the œsophagus in the mediastinum. Attached to that part of the stomach which comes away are the left half or third of the great omentum, spleen, body of pancreas, and retroperitoneal tissues as for

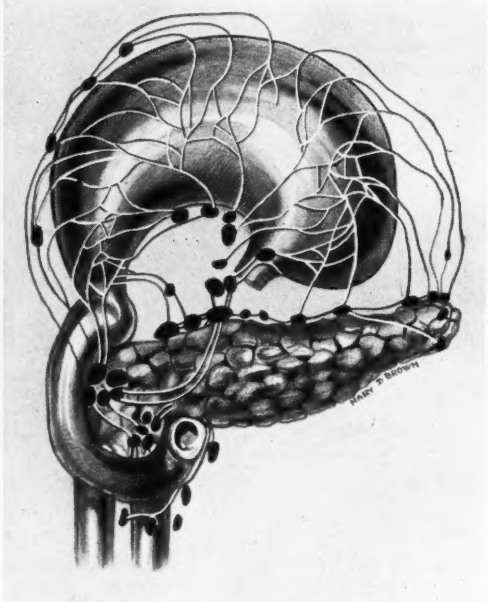


FIG. 11.—Lymphatics of the stomach as seen when stomach is rotated upwards to expose its posterior surface. (*After Jamieson and Dobson.*)

carcinoma of the stomach. More mediastinal tissue is taken with the œsophagus and strips of both mediastinal pleuræ if necessary. If the growth has invaded the stomach it is probably better to remove the whole stomach and anastomose the œsophagus to the jejunum.

Selection of Patients for Radical Operation.—The patients are selected mainly by a process of elimination. The clinical findings which make patients unsuitable for the radical operation are: first, distant metastases, particularly in the glands at the root of the neck, in the liver, umbilicus, or pelvic peritoneum; secondly, gross arterial degeneration, for the security of a mediastinal anastomosis depends upon elastic arteries; and thirdly, poor cardio-respiratory function, for lung infections are particularly liable to occur after operation, and if the patient has no cardio-respiratory reserve he soon develops a fatal bronchopneumonia. Thin patients as a rule are better risks than fat ones. Although loss of weight due to poor feeding does not preclude operation after suitable preparation, there seems to be a stage of emaciation with this disease beyond which resistance to operation, resistance to sepsis, and sound healing of wounds cannot be restored.

Pre-operative Preparation.—The patient is usually kept in hospital for about two weeks before operation, during which time he is given a high-calorie fluid and semi-solid diet with extra vitamins. In the absence of obstruction caused by solid food this diet is usually taken well, but if there is difficulty

the stricture is gently relieved by the passage of bougies through the œsophagoscope. Even if solid food can be swallowed it is not allowed for the two weeks before operation in order to reduce stagnation and sepsis. The gullet is washed after each meal by a drink of water or soda-water. If it is felt that the patient will be fit for operation and he has gross dental sepsis the teeth are removed under sodium pentothal anæsthesia at the initial diagnostic œsophagoscopy, so that two weeks are allowed for the gums to heal. It has not been thought necessary to remove teeth as a routine, but during his stay in hospital the patient's mouth is made as clean as possible by tooth brushing and mouth-washes. Feeding by mouth is supplemented by intravenous plasma if there has been much loss of weight and a slow blood-drip of two or three pints is given about a week before operation. There are many biochemical changes associated with partial starvation which are not understood, but there seems to be little doubt from clinical observations that these patients feel better and are better operative risks after blood transfusion, even though no abnormality in the blood-picture can be detected. This transfusion is in addition to the routine drip used during the course of operation. An exploratory abdominal operation with a view to gaining further details about the growth, doing a palliative gastrostomy if inoperable or a preliminary feeding gastrostomy if operable, is condemned. The disadvantages of palliative gastrostomy have already been mentioned. Preliminary gastrostomy is unnecessary, for better results can be obtained by intravenous and oral feeding after careful radiography and œsophagoscopy. The final decision as to operability is best left to the set operation, when an exposure is made through which both ends of the growth can be explored and through which either radical or palliative surgery can be carried out. Apart from these considerations it is fundamental that the burden of treatment should not be increased beyond what an elderly person can be expected to bear cheerfully, and for this reason two operations should not be used when one will do as well. For the same reason it is most desirable that transfusions should be given with a needle and with dexterity, that œsophagoscopy should be free from trauma, and that dental extraction should only be done when the teeth and gums are beyond other treatment. An effort is made to mobilize the chest and increase respiratory excursion by a trained physiotherapist, who visits the patient once or twice a day during his stay in hospital; and, although the wind is tempered to the shorn lamb, notable improvement in chest and abdominal movements can usually be observed. Smoking is forbidden during this two weeks, but the necessity for this is carefully explained. The patient is assured that he can resume smoking immediately after operation. An intramuscular injection of one mega-unit of penicillin is given the night before operation and a second in the morning.

Anæsthesia.—Morphine gr. $\frac{1}{4}$ and scopolamine gr. $\frac{1}{150}$ are given and a blood-drip is started. Enough tubarine and sodium pentothal are run into the drip to relax the jaw and the œsophagoscope is passed. Great care is taken to aspirate the œsophagus dry and clean. Immediately above the obstruction the aspirator is abandoned and swabs used to dry out this part of the gullet so that there is no trauma to cause bleeding from the growth. A little penicillin powder may be blown in, and as the œsophagoscope is withdrawn a 'Tampax' pack is inserted through it to lie in the gullet just below the cricoid. With this technique it is reasonably certain that there will be no reflux of septic fluid from the growth or from the œsophagus itself into the pharynx which could be inhaled during the early stages of the operation. The nasopharynx is then cleaned out by passing a well-greased No. 10 Jacques rubber catheter through each nostril and applying suction. It has often happened that 2 or 3 c.c. of purulent mucous secretion has been removed in this way. Such a pool would be a source of danger during the manipulation involved in placing the unconscious patient in position on the table and also during the immediate post-operative phase. A pharyngeal airway is then introduced, and, as a further precaution against inhalation of secretions, a light pack is inserted around this. When a well-fitting face mask is applied inhalation of oxygen or anæsthetic agents can occur without fear of septic aspiration and without the irritation which is inevitable when an intratracheal tube is in position for four or five hours. The patient is then turned on to his right side and fixed in the position for operation with a pillow under his waist. He should incline neither forwards nor backwards so that access to abdomen and chest is free. If he is considered to be a good surgical risk he is given a spinal anæsthetic of light percaine, but if the general condition is less favourable this is replaced by blocking the lowest left eight intercostal nerves with local anæsthetic. The latissimus dorsi is also infiltrated with local anæsthetic at the level of the ninth rib. The left splanchnics, solar plexus, the vagi, and the left phrenic nerve are anæsthetized under direct vision as soon as the chest is opened. The solution used for infiltration is :—

Procaine	0·25 per cent
Sod. chlor.	0·89 per cent
Amethocaine hydrochloride	0·05 per cent

to which adrenaline is added immediately before use in the proportion of 10 minims of 0·001 per cent adrenaline to 250 c.c. of solution.

With this combination of agents the first part of the operation can be completed with oxygen only being inhaled. In the latter stages, however, the patient may become more sensitive and a little restless. It has been the custom in the past to overcome this by further injections of pentothal, but it is now thought to be a disadvantage to do this as the patient remains unconscious far too long after return to bed. For this reason, therefore, cyclopropane may be used if necessary during the last hour or so of the operation. During much of the surgical manipulation both lungs can be kept normally inflated, but when dissection in the mediastinum requires lung retraction small areas of absorption collapse are almost sure to occur. These are not harmful unless left too long. The anæsthetist should be given an opportunity to inflate them at least every twenty minutes. This should be done both gently and slowly to avoid rupture of alveolar walls. At the end of the operation complete inflation of the lungs is secured whilst the chest is still open and

this is maintained until the chest is air-tight except for a sealed underwater drain. Before the position of the patient is altered the pack is removed from the pharynx and the nasopharynx again aspirated through a soft rubber catheter inserted through each nostril in turn. During an anæsthesia lasting about four hours a moderate amount of mucus may collect in the nasopharynx. Even if this is not mixed with mucopus it is always heavily infected, and could easily be aspirated into the trachea during the movement of the patient on to his back or whilst he is still unconscious in his bed. Only after this careful toilet is the patient turned on to his back. This should be done slowly to avoid a sudden drop in blood-pressure. The ' Tampax ' pack is removed from the upper end of the œsophagus by traction on its silk thread over a finger inserted in the pharynx. Unless the finger is used as a sort of pulley the silk thread will drag over the larynx and injure it. The œsophagoscope is immediately passed and any fluid or blood-clot aspirated from the gullet. The anastomosis can be inspected at the same time. The chest is X-rayed in the theatre to make sure that both lungs are fully expanded.

Continuous oxygen is given by nasal catheters after the patient's return to the ward and the blood-drip is kept going slowly. The underwater drain is removed after about 24 hours. After a long operation of this magnitude the patient recovers semi-consciousness and many of his reflexes return a long time before he complains of any pain. Sedatives are withheld as long as possible to avoid stagnation in the respiratory tract, but at night time on the day of operation he is given $\frac{1}{4}$ gr. morphine. After this it is administered $\frac{1}{8}$ gr. at 4–6 hourly intervals as required, as this alleviates pain enough to permit deep breathing without depressing the respiratory centre.

Operation.—The full thoraco-abdominal incision starts from the middle of the left rectus abdominis muscle and sweeps backwards to cross the costal margin at the tip of the ninth costal cartilage. It follows the ninth rib back to the outer border of the erector spinæ muscle, where it curves upwards for 2 in. After the skin incision has been made the muscles are divided with the diathermy. The posterior end of the eighth rib and the whole of the ninth rib are removed. At the anterior end the costal cartilage is dissected away from the muscles with the diathermy knife. The pleura and peritoneum are opened. The diaphragm is split back towards the hiatus from the anterior end of the pleural incision. As the vessels in the diaphragm are caught they are stitched. It is convenient to use these stitches as retractors, the anterior ones being sewn to the external oblique muscle on the edges of the incision, the posterior ones being left long and anchored to the surface by artery forceps (*Fig.* 12). The fixation of the diaphragm in this way not only makes retractors unnecessary, but allows proper action of the right dome of the diaphragm and prevents the pull of this from kinking the inferior vena cava and obstructing the blood return to the heart. When this incision has been completed there is no need for retractors, for the edges fall apart and there is ample room to do both the abdominal and thoracic dissections. The mediastinal pleura is incised at the level at which the œsophagus is to be divided. The vagus nerves are divided and the œsophagus drawn out from its bed. A thin anastomosis clamp is then applied to the œsophagus just below the point chosen for section and firmly closed, so that there is no possibility of infected or irritating fluids escaping upwards during the mobilization of the stomach. It has been thought desirable to choose the level for division of the œsophagus at least 2 in. above the top of the growth. Emphasis has already been laid on the need for noting the highest point involved by the most careful œsophagoscopic examination rather than relying entirely on external palpation at the time of exposure. The

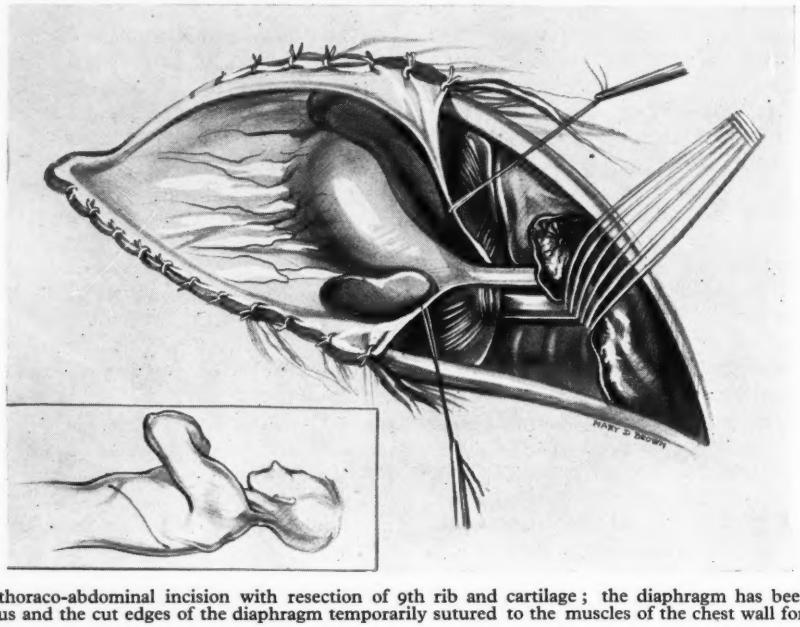


FIG. 12.—Left thoraco-abdominal incision with resection of 9th rib and cartilage; the diaphragm has been split back into the hiatus and the cut edges of the diaphragm temporarily sutured to the muscles of the chest wall for fixation.

technique from this time varies according to whether a partial or a total gastrectomy is to be performed.

Total Gastrectomy.—It is an advantage to fashion the intestinal loop as the first step in the abdominal part of the operation, for the division of the vessels in the mesentery according to the method of Roux is both delicate and tedious and it is, therefore, better done before the surgeon is tired. It has a further advantage in that the loop of intestine, when made the required length, may be dropped back into the abdomen until the last stage of the operation. There can then be no doubt about its viability when the final anastomosis is to be performed. Intestinal continuity is restored by end-to-side anastomosis at the base of the loop, and the cut edge of the mesentery of the upper jejunal segment is stitched to the right side of the mesentery of the loop to prevent internal hernia. The site of intestinal anastomosis is carefully chosen to avoid drag on the loop or kinking of the upper jejunal segment. It is performed without clamps so that no damage to the veins in the mesentery of the loop is possible. Where the free edge of the mesentery of the loop is likely to lie across the duodenojejunal flexure the peritoneum around the latter is divided and the flexure displaced.

The spleen is drawn forwards and the outer layer of the lienorenal ligament divided. The division of this layer of peritoneum is carried up over the diaphragm nearly to the hiatus. Traction on the spleen and a combination of sharp and blunt dissection then displaces the whole stomach bed forwards. If the separation is easy the suprarenal capsule is left behind, but if there is fixation here the capsule may be taken forwards with the spleen, splenic vessels, and pancreas. The left crus of the diaphragn where it is fanning out is cleaned and divided into the hiatus after ligature and division of the inferior phrenic vessels. The sustentaculum lienis is divided and the lesser sac opened below the spleen. The great omentum is drawn up and separated from the transverse colon as far as the first part of the duodenum. As the pancreas is drawn forwards the peritoneal reflection from the lower border is divided until the inferior mesenteric vein is reached (*Fig.* 13). The fold of peritoneum between the pyloric antrum and the pancreas is divided and the duodenum freed as far as the pancreatico-duodenal vessels which lie on its posterior surface. The right gastro-epiploic vessels are divided at their junction with the parent vessels. The upper border of the pylorus and adjacent part of duodenum is freed by division of the pyloric vessels and a crushing clamp applied across the duodenum. With the mass of tissue still displaced forwards the splenic vein is isolated where the inferior mesenteric vein joins it and it is divided between ligatures so as to leave the tributary from the large intestine intact. The splenic artery is ligatured and divided where it arises from the cœliac axis and at this stage it may be necessary to divide the left gastric vein where it crosses to join either the splenic or portal vein. The pancreas is divided with the diathermy knife and a few bleeding vessels in its substance secured with a running stitch. The stomach is still held to the posterior abdominal wall by the left gastric artery, and some cellular and lymphatic tissue. The vessel is cleaned and divided at its origin and the surrounding lymphatic and nerve tissue which often contains small veins ligatured and divided. The mass of tissue is now drawn back, and the lesser omentum divided from the top of the duodenum along the hepatic artery and portal vein to the hilum of the liver and along its attachment to the liver, where a few small vessels should be ligatured. This brings the dissection up to the right side of the hiatus of the diaphragm. The peritoneum here is incised, and the right inferior phrenic vessels and the fibres of the right crus which form the hiatus are divided. The cellular tissue immediately above the hiatus is drawn away from the diaphragm, pericardium, and right pleura, and the dissection carried up into the mediastinum as

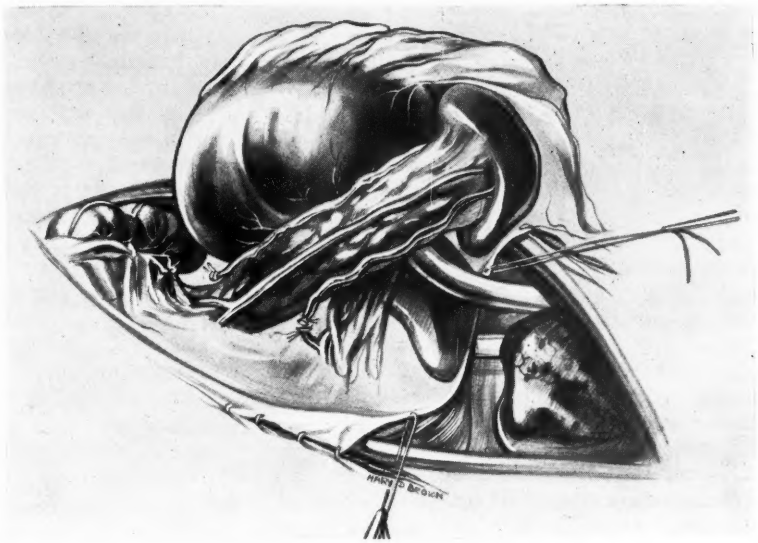


FIG. 13.—Stomach and great omentum, spleen, and pancreas drawn forwards and peritoneum from lower border of pancreas divided as far as the inferior mesenteric vein.

far as the clamp on the œsophagus. The growth and surrounding mass of tissue is thus completely freed except for its continuity with the œsophagus above and the pylorus below. A second clamp is placed across the pylorus, and the duodenum divided and sutured with infolding. A second light anastomosis clamp is applied to the œsophagus above the first one and the œsophagus divided. The mass of tissue is then removed. The jejunal loop is threaded through a hole in the transverse mesocolon and laid in the mediastinum to make sure that it reaches the œsophagus without tension (*Fig.* 14). The place for anastomosis on the anterior aspect of the upper end of the intestinal loop is noted. The loop may then be withdrawn from the chest and an incision made along the line of anastomosis through the peritoneal and muscular coat down to the submucosa. If this is done with the intestine outside the chest it is easy to understitch and divide the main vessels in the intestinal wall and so avoid the choice between the use of clamps or the constant oozing of blood into the field of anastomosis. The loop is replaced in the mediastinum and a row of interrupted catgut stitches inserted through its peritoneo-muscular wall above the incision and the fibromuscular wall of the œsophagus. Its lumen is then opened. A single holding suture is passed through the middle of the anterior wall of the œsophagus to include the muscle and mucous membrane and maintain them in contact. This stitch is left long and a clip attached to it outside the wound. A continuous catgut stitch is then inserted through the full thickness of jejunum and œsophagus to unite their posterior walls. This stitch starts at the far end of the anastomosis and is taken around the near end to the middle of the anterior walls, where it is left long and clipped. A second continuous stitch starts at the same place as the first to which it is tied, and passes along the anterior wall towards the middle line (*Fig.* 15). It is tied to the first one in the middle of the anterior walls. At this time the clamp is released from the œsophagus so that the anterior and posterior walls separate. This avoids the possibility of stitching the anterior and posterior walls together. Traction on the ' holding stitch ' in the œsophagus ensures that the mucous membrane of the œsophagus does not slip upwards and escape from the continuous stitch as the anastomosis is completed. It is better to finish the continuous full-thickness stitch in front, where it is clearly visible, than at either end, where a small section of either wall might escape. A single row of interrupted catgut stitches is placed anteriorly through the fibromuscular and peritoneo-muscular walls to sink the line of junction of the mucous membrane. This layer corresponds to the first row inserted at the back and completes the anastomosis. The loop is loosely sutured to the mediastinal pleura and to the edge of the diaphragm by a few interrupted stitches. The diaphragm is unpicked from the external oblique muscle to which it has been sutured and is closed around the loop and its mesentery. A little penicillin and sulphonamide powder is dusted into the subphrenic spaces and mediastinum and the chest closed in layers with an underwater drain in the pleura. The drain is removed after 12 to 24 hours when radiography shows all the air to have been expelled from the abdomen and pleura.

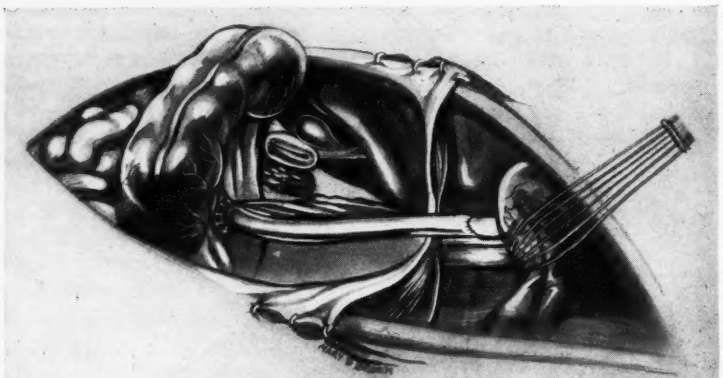


FIG. 14.—Jejunal loop threaded through transverse mesocolon and passing up into mediastinum to the cut œsophagus.

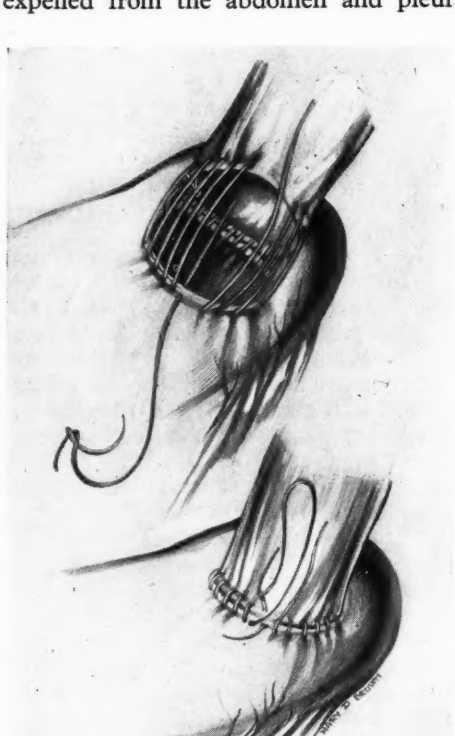


FIG. 15.—Continuous full-thickness suture so arranged as to finish at about the middle of the front of the anastomosis.

Partial Œsophago-gastrectomy.—Where the location of the growth justifies only partial resection of the stomach, the aim should be to remove the cardia, fundus, and practically the whole of the lesser curvature, leaving the greater curvature and adjacent part of the body of the stomach to form a tube which can be anastomosed to the œsophagus. No jejunal loop is therefore necessary. The spleen and

pancreas and retroperitoneal tissues are displaced forwards as already described. Only the left half of the great omentum is removed with the fundus of the stomach. The dissection along the greater curvature of the stomach is not taken as far as the pylorus, but four or five of the branches of the right gastro-epiploic vessels are left intact. The pancreas and splenic vessels are divided as above. The pyloric vessels are not divided, but a point is chosen on the lesser curvature of the stomach where they anastomose with the left gastric vessels. The lesser omentum is divided here and the adjacent part of the curvature cleared of vessels. Clamps are then applied obliquely from this position across the stomach to the point on the greater curvature which has been cleared of great omentum. The stomach is divided and closed and the mass of tissue containing the growth removed. As this operation is usually done for an œsophageal carcinoma, the resection of the œsophagus is correspondingly higher than when total gastrectomy is done. The remaining part of the stomach is swung up into the mediastinum and joined to the cut end of the œsophagus in the same way as already described for the jejunum.

The operation technique has been developed gradually during the last six years and no great numbers have yet been done by a standard method. Relatively recently it has been decided that the treatment for a primary gastric growth should be total gastrectomy, the partial operation being performed for the low œsophageal growths. The incidence of post-operative œsophagitis and peptic ulcer after œsophago-gastrectomy, however, may lead to the adoption of the bigger operation with œsophago-jejunostomy in most cases.

Post-Operative Treatment.—The patient is nursed flat in bed after operation and is given sips of fluid as may be required from the beginning. Parenteral fluids are continued until intestinal movements can be heard, usually 48 hours after operation, when they are replaced by a normal intake of fluids by mouth. A basal calorie intake can usually be taken and absorbed on the third day in the form of glucose-water, soup, tea, coffee, and milk. On the fifth day soft solids are given, bread and butter, eggs, potatoes and gravy, and by the tenth day the patient should be taking a normal diet. Although he is kept flat in bed during the first twelve hours he is allowed to sit up and move about as soon as he is able. Chest exercises with assistance at coughing and leg exercises are given by the physiotherapist as soon as consciousness returns. He is encouraged to get out of bed. Usually on the third day he sits out while the bed is made, and he should have tea in his chair on the fifth day. After partial resection with œsophago-gastrectomy the patient learns to sleep propped up in bed to diminish reflux of acid into the stomach, and after total resection this is probably a wise prophylactic measure to avoid inhalation of intestinal contents during deep sleep. In the latter case the diet should be supplemented by iron and liver extract.

Complications.—Local sepsis in the mediastinum or pleuræ or peritoneum may be fatal in the first three days after operation, especially where infected fluid from a sloughing growth has been allowed to escape during operation. This is minimized by most careful pre-operative toilet of the œsophagus before operation, intramuscular penicillin starting the day before operation, avoidance of spill, and the local application of penicillin and sulphonamide powder to the operation area. Full expansion of the lungs is important for localizing sepsis if it occurs. Empyema or subphrenic abscess may develop later and call for drainage.

Mild bronchitis is not uncommon, but bronchopneumonia is usually fatal. There may be many factors in the production of bronchopneumonia, but the assumption that it is always due to the inhalation of septic secretions during and immediately after operation has the advantage that it is very nearly true and that it is a factor which can be eliminated if enough care is used. If bronchitis is present beforehand the operative risks are of course greatly increased. Massive pulmonary collapse only occurs as a complication of infection in the bronchial tree and should be treated by intensive physiotherapy. If the lung is not expanding within three hours of this treatment bronchoscopic aspiration should not be delayed.

Cardiac or peripheral vascular failure if they occur are likely to be seen in the first 48 hours, but as many of these patients have a poor cardiovascular system sudden fatalities may happen later. Although good lungs and good arteries make for good results, patients who have previously had coronary attacks or auricular fibrillation have survived the radical operation.

Some degree of intestinal ileus is usually noted for 48 hours after operation. It should recover spontaneously and no effort is made to stimulate the bowel. Rest with small doses of morphine tide the patient over this phase. In one instance damage to the inferior mesenteric vein was associated with rather alarming distension of the colon for ten days, but this, too, gradually settled without active treatment. There may be some delay in the healing of wounds where a carcinoma has been present for long and nutrition has been impaired, but the routine use of blood transfusion appears to have reduced the incidence of the complication. Some circumspection is necessary, however, in deciding when stitches and dressings are to be removed. A leaking anastomosis in the first three days is almost certainly caused by technical imperfection. Where it has occurred in this series it has always proved fatal from pleurisy and peritonitis. A fistula appearing a week or later after operation is caused by tissue necrosis and poor vascularity of the stomach or intestine. The œsophagus itself, contrary to the old teaching, remains viable after much mobilization and has never shown any sign of leaking in these patients. Where the stomach has been joined to the œsophagus and fistula has occurred a post-mortem has shown sloughing of a large part of the stomach. These patients have been arteriosclerotic and the cœliac axis has been the seat of notable atheroma. In one patient the sloughing led to acid digestion of the aorta with sudden and fatal hæmorrhage. When a jejunal loop becomes gangrenous from thrombosis of the vessels in its mesentery the picture appears to be well defined. The patient remains well for 24 to 48 hours after operation. Quite suddenly the

pulse-rate rises to about 120 and is of low volume. The skin becomes cold and moist, the lips slightly blue, and the face anxious. Death occurs in six to ten hours.

Of the later complications the most important are recurrence of the growth and, where œsophago-gastrostomy has been performed, œsophagitis, peptic ulceration, and sometimes stenosis. The recurrence of carcinoma in the stomach itself has already led to the adoption of total gastrectomy in all cases of operable gastric growths. Partial resection for œsophageal carcinoma is still under review because of the unpleasant sequelæ of peptic digestion of the œsophagus, but it is not yet possible to condemn the operation. Attacks of diarrhœa can usually be avoided by careful mastication and small meals, but if this is not always enough the patient should take a mixture containing three minims of tincture of opium with each meal until the attack has passed. Anæmia should be prevented by the taking of liver extract permanently.

Although no case of steatorrhœa has been observed in this series, it has occurred in a patient treated more recently. There seem to be two possible explanations for its occasional occurrence: first, removal of the body of the pancreas; and, secondly, the too rapid passage of food through the small intestine before the pancreatic juice, passing through the duodenum and upper jejunum to the T-anastomosis, can catch up with it. In the one patient in whom this complication has appeared it has been treated by the administration of a little soup ten minutes before the meal in order "to give the pancreatic juice a start". There has been a gradual improvement over the course of a few months.

RESULTS

Of the 310 patients seen, medical treatment or intubation was used in 189 (60 per cent). These patients were hopeless when first seen either because of the extent of the growth or from associated disease. In spite of this the selection for operation has been very generous and treatment has been assessed on the basis of improving the patient's lot rather than the mortality figures. Of the 121 cases subjected to operation, 13 were done before June, 1942, and were mostly local excisions with ante-thoracic skin œsophagoplasties. One of these patients lived for over four years, to die of heart failure with no post-mortem signs of recurrence. Since that time resections with mediastinal anastomosis have been done and the following figures refer to these. Since June, 1942, 108 operations have been done, of which:—

25 were exploratory or miscellaneous,
10 palliative œsophago-jejunostomies without excision of the growth,
49 partial resections, and
24 total gastrectomies with partial œsophagectomy

All the resections were accompanied by radical lymphatic dissections except where lymphatic spread was so extensive that only palliative resection of the primary growth was feasible.

The 25 patients in the first group and the 10 in the second are all dead. Of the 49 in whom partial œsophago-gastrectomy was performed, 26 (53 per cent) died from the effects of the operation and 23 (47 per cent) survived, and in only 13 (27 per cent) was convalescence completely uncomplicated. Operative deaths were as follows:—

Bronchopneumonia	7
Pleural or peritoneal infection	4
Pulmonary embolus	3
Cardiorenal failure	4
Volvulus of stomach on its long axis (anastomosis above aortic arch)	1
Strangulated diaphragmatic hernia	1
Leaking anastomosis	1
Sloughing of upper part of stomach	4
Purpura	1

Where the stomach sloughed from poor vascularity death usually occurred between the ninth and fourteenth day after operation, and at autopsy gross atheroma of the cœliac axis was found. In one of these patients the necrosis of the stomach left a gap, in the floor of which was the aorta. Death followed acid erosion of the aorta with massive hæmorrhage. Of the 23 patients who survived operation 9 are still alive and 14 are dead.

SURVIVAL TIMES FROM DATE OF OPERATION

Already Dead	*Still Alive (June, 1948)*
3 months	7 months
6 months	1 year
6 months	1 year
6 months	1 year 1 month
7 months	1 year 7 months
10 months	2 years
1 year	2 years 2 months
1 year	3 years
1 year 1 month	3 years 3 months
1 year 3 months	
1 year 4 months	
1 year 6 months	
2 years 5 months	
2 years 6 months	

It must be remembered that many of these operations were performed when the growth was far advanced and that in some palliative resection of the tumour was performed where secondary deposits made a radical excision impossible.

Of the 24 patients for whom total gastrectomy was performed, 7 died from operation, a mortality of 29 per cent. The causes of death were as follows:—

Bleeding from pancreas	1
Bronchopneumonia	1
Leaking anastomosis	1
Peritonitis and pleurisy	1
Cardiorenal failure	1
Thrombotic gangrene of leg and bronchopneumonia	1
Auricular fibrillation and myocardial insufficiency (2 months after operation)	1

Bleeding from the pancreas and leaking anastomosis were technical faults which should not have been allowed to occur and this may also be so for the one example of pleurisy and peritonitis. It has been noted, however, that the incidence of septic complications is higher where histological examination of the tumour shows evidence of suppuration within it; and it may therefore happen that post-operative sepsis is not necessarily a reflection on technique. The other complications causing death are some indication of the physical state of many of the patients treated. In addition to the operative deaths, 3 patients have already died from metastases 52 days, 5 months, and 7 months after operation. There were therefore in June, 1948, 14 patients alive, and the following is their span in months since operation: 2, 2, 2, 2½, 3, 4, 4, 6, 9, 12, 12, 13, 15, 25.

The post-operative complications included pulmonary collapse, empyema, subphrenic abscess, intestinal obstruction, ileus, wound infection, bedsore, strangulated femoral hernia, phlebitis of legs, and diarrhœa, in addition to those from which patients died.

THE EXAMINATION OF LYMPHATIC GLANDS AFTER RADICAL BLOCK DISSECTION FOR CARCINOMA OF THE STOMACH AND LOWER END OF ŒSOPHAGUS

The appreciation of the lymphatic anatomy of the stomach as described by Jamieson and Dobson in 1907, combined with improved anæsthesia, blood transfusion, and the many other factors which make a thoraco-abdominal approach and radical dissection possible, have, for the first time, provided pathological specimens which could give a clear picture of the spread of malignant disease of the stomach and lower œsophagus. The results so far obtained are not comprehensive enough for any wide classification and the ultimate answers will only be found after many years' study of specimens and survival rates. Enough has already been done, however, to indicate that malignant cells do follow the anatomical pathways which Jamieson and Dobson described, that the radical removal of the tumour with its lymphatic bed is feasible without a prohibitive mortality, and that those patients who recover may be capable of normal activities and that there is not as yet any way of forecasting which tumours have spread to which lymph-nodes.

There is, therefore, theoretical justification for applying the radical operation to all operable tumours and a reasonable hope that patients with early disease treated in this way may have the expectation of a longer survival than has been their lot with either partial or total gastrectomy.

Procedure.—Since July, 1947, all operation specimens of carcinoma of the œsophagus and cardia have been carefully dissected, each lymphatic gland being sectioned and examined separately.

At the conclusion of a total gastrectomy for carcinoma of the cardia the specimen is inflated with 10 per cent formalin solution. Following a partial œsophago-gastrectomy for carcinoma of the lower third of the œsophagus the specimen is packed with formalin-impregnated cotton-wool, and later immersed in 10 per cent formalin solution. A careful outline drawing of it is made at once, and the glands are dissected in the fresh state. As each is removed, so its size and site are charted on the drawing. This dissection may take over an hour to complete. So far the numbers removed from any specimen have ranged from 6 to 43 glands.

The glands are numbered to correspond with the chart and threaded on to nylon. To save the number of paraffin blocks several glands, usually 3–4 depending on size, are sectioned together. The preparing of slides of these glands is the most time-consuming step in this study, requiring the watchful care of an enthusiastic technician. When fixed, the specimen itself is cut, re-coloured, painted, and an outline diagram made on transparent celluloid, similar to the original chart.

Table I.—Pathology Investigation

Serial No.	Case No.	Type of Growth	Direct Invasion		Lymph-glands	The Sites of the Glands							
			Soft Tissue	*Other Organs*	*No. Dissected*	*No. Involved*	*L. Gastric*	*Para-cardial*	*Splenic*	*L. Gastro-epiploic*	*Supra-pancreatic*	*Sub-pyloric*	*Post-mediastinal*	*Great Omentum*
Radical Total Gastrectomies with Partial Œsophagectomy.—														
a	1	Adenocarcinoma	Yes		33	14	3			2				1 + 8 metastases
b	3	Adenocarcinoma	Yes	Left lobe of liver	22	17			1	2	2	4	5	3
c	4	Adenocarcinoma	Yes		14	1	1							
d	5	Adenocarcinoma	Yes	Left suprarenal gland	43	10	8	2						
e	6	Adenocarcinoma	No		29	1	1							
f	10	Adenocarcinoma	Yes		33	23	10	4			1		8	
g	11	Adenocarcinoma	Yes		18									
h	12	Squamous carcinoma	Yes		15	1	1							
i	13	Adenocarcinoma	Yes		9	2	1	1						
j	15	Adenocarcinoma	Yes		30	10					3			1
Radical Partial Œsophago-gastrectomy.—														
a	2	Squamous carcinoma	Yes		21									
b	7	Adenocarcinoma	Yes		6	4	2						2	
c	8	Squamous carcinoma	Yes		13	9	5	1					3	
d	9	Squamous carcinoma	Yes		21	4	3				1			
e	14	Adenocarcinoma	Yes		3	2	1	1						

The primary growth and glands have been examined in the Department of Experimental Pathology, reports on the extent of invasion being made by Professor M. J. Stewart and Professor J. Orr. So far, it has been considered sufficient to complete one section of each gland. The blocks, however, are kept so that a 'negative' report from a suspicious area can be re-checked by cutting serial sections. When these results are known, the celluloid charts are completed. If invaded, the glands are blacked out on the charts. If not invaded, the centres are left clear. Photostat copies and slides of these charts are easily made.

Results.—From July, 1947, to July, 1948, 15 specimens have been thus investigated. No patient was seen so early that the growth was confined entirely to the wall of the viscus. Either direct invasion into the surrounding tissue or spread to the regional lymph-nodes had already occurred. In only 1 patient (*Case* 6) had the primary growth not breached the muscle wall, and in this distant metastasis was present. Only 2 cases showed no metastasis to the regional lymph-nodes (*Cases* 2, 11), yet in both the growth had breached the muscle wall. Three cases (*Cases* 4, 6, and 12) had only one glandular metastasis, on each occasion being situated in the left gastric group of lymph-nodes. In this series the left gastric group of lymph-nodes was always the earliest involved. In five patients pancreatic glands were invaded, *Cases* 3, 9, 10, 15 showing involvement of the suprapancreatic group and *Case* 5 the infrapancreatic group. In others, scattered glands in the cellular tissue between the fundus of the stomach and the upper border of the pancreas were invaded.

The findings are summarized in *Table I.*

If we adopt for carcinoma of the stomach and lower œsophagus a classification similar to that suggested from St. Mark's Hospital for carcinoma of the rectum, four stages in the progress of the disease may be recognized: growth limited to the œsophagus or stomach in Stage I; growth spreading by direct continuity into the adjoining structures, but not giving rise to lymphatic metastases in Stage II; further spread, with lymphatic metastases in Stage III; spread beyond the scope of surgical cure in Stage IV.

In this series the results were :—

Stage	I	Nil
Stage	II	2 (*Cases* 2, 11)
Stage	III	10 (*Cases* 4, 5, 6, 7, 8, 9, 12, 13, 14, and 15)
Stage	IV	3 (*Cases* 1, 3, and 10)

Another striking finding was the way in which the neoplasm readily burrowed up under the normal mucosa in the submucous layer. This was seen in seven patients (*Cases* 1, 7, 8, 9, 13, 14, 15). This emphasizes the necessity for resecting the œsophagus as high as possible.

One has been impressed by the large number of glands thought to be invaded which microscopically showed only catarrhal inflammation. In *Case* 5, 43 glands were dissected but only 10 invaded. With carcinomatous ulcers at this site, therefore, more glands are enlarged by infection from ulcer than by invasion from cancer.

SUMMARY

1. Experiences with the treatment of 310 patients suffering from malignant obstruction of the cardia are described.

2. Whilst palliative surgery has much to offer these patients, a case is stated for a more logical approach to the operable growth based on the observed direct spread of cancer in this region and on the lymphatic researches of Jamieson and Dobson.

3. A suggestion is made that carcinoma of the stomach should be treated by total gastrectomy with block dissection of the lymphatic bed and that this inevitably entails resection of the spleen and body of the pancreas. Carcinoma of the lower œsophagus requires the same block dissection but alimentary continuity may be restored either by œsophago-gastrostomy or œsophago-jejunostomy.

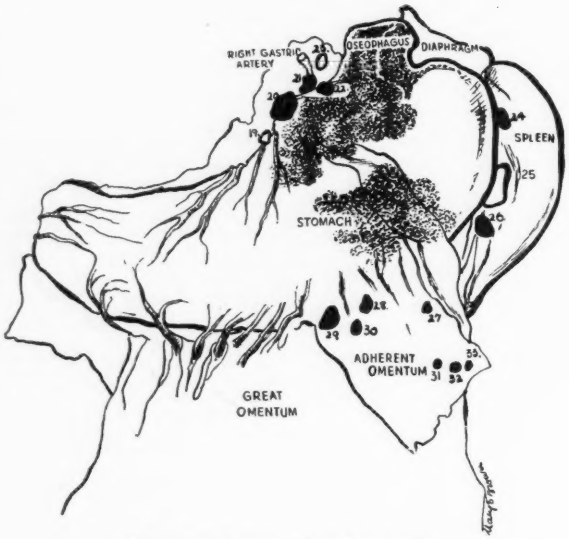


FIG. 16.—*Case* 1. Anterior aspect.

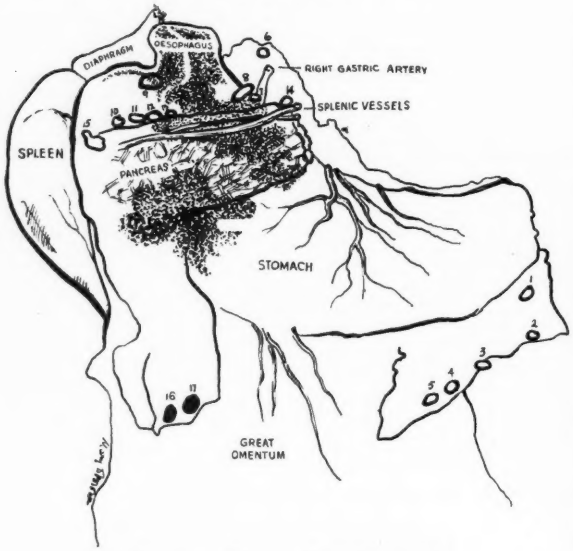


FIG. 17.—*Case* 1. Posterior aspect.

Key to *Figs.* 16–44: Extent of growth Glands invaded by growth

4. Post-operative dissection of lymph-glands and accurate charts have been made in 15 patients suffering from carcinoma of the cardia. Each gland was histologized. The method is described, and the charts are published. Four stages of spread are recognized :—

a. Growth limited to the œsophagus or stomach —Stage I. (No cases were seen in this favourable stage.)

b. Spread by direct continuity into the adjoining structures without lymphatic metastases—Stage II. (There were 2 patients in this group.)

c. Further spread with lymphatic metastases—Stage III. (There were 10 patients in this group.)

d. Spread beyond the scope of surgical cure—Stage IV. (There were 3 patients in this group.)

The left gastric group of lymph-glands was the earliest and most extensively invaded. Glands around the body of the pancreas were invaded in 5 cases. The neoplasm readily spread up the œsophagus in the submucosa, under an apparently normal epithelium. It was noticed that more glands were enlarged by infection from the ulcer than by invasion from the cancer.

CASE REPORTS

Case 1.—R. H. (64), *carcinoma of stomach* (*Figs.* 16, 17).

July 10, 1947 : Total gastrectomy, splenectomy, omentectomy. Hemi-pancreatectomy and partial œsophagectomy. Retrocolic Roux loop reconstruction.

Nov. 15 : Re-admitted with liver metastasis.

Feb. 6, 1948 : Died.

Pathology Summary : An ulcerating adenocarcinoma with focal suppuration. The stomach wall is extensively breached, and the gastrocolic omentum invaded. At its upper end the tumour has extended into the œsophageal wall under the squamous epithelium, which is intact.

Metastases : In 7 lymph-glands. Nodules 24, 27–33 are also metastatic deposits ; but are not lymph-glands. 33 pieces of tissue dissected and examined.

Case 2.—Mrs. M. B. (68), *carcinoma of lower third of œsophagus* (*Fig.* 18).

July 29, 1947 : Partial œsophagectomy, partial gastrectomy and omentectomy, hemi-pancreatectomy, and splenectomy.

Aug. 15 : Died from massive pulmonary embolus.

Pathology Summary : An exceedingly invasive squamous carcinoma which has breached the œsophageal wall and invaded the surrounding cellular tissue.

Metastases : In no glands. 21 examined.

FIG. 18.—*Case* 2.

Case 3.—F. G. (58), *carcinoma of stomach* (*Figs.* 19, 20).

Aug. 19, 1947 : Total gastrectomy, splenectomy, omentectomy, hemi-pancreatectomy, and excision of

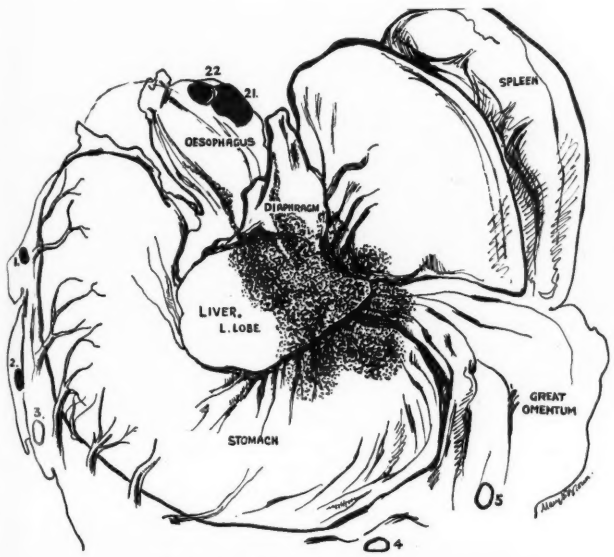


FIG. 19.—*Case* 3. Anterior aspect.

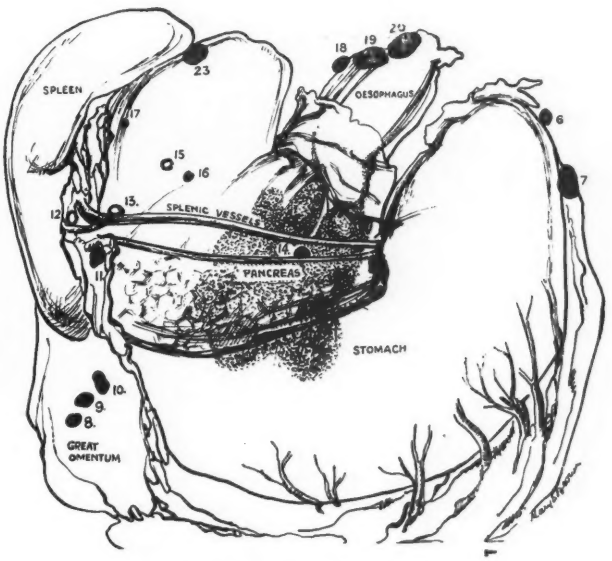


FIG. 20.—*Case* 3. Posterior aspect.

lower third of œsophagus. Retrocolic Roux loop reconstructed. Portion of left lobe of liver removed.

Aug. 22: Died. Bronchopneumonia.

Pathology Summary: A massive carcinoma of encephaloid type encircling the cardia and extending into the œsophagus. 7 cm. in diameter. 3 cm. thick. Direct invasion of left lobe of liver.

Metastases: In 17 glands. 22 pieces of tissue dissected. 21 examined. (No. 4 not a lymph-gland; No. 5 lost.)

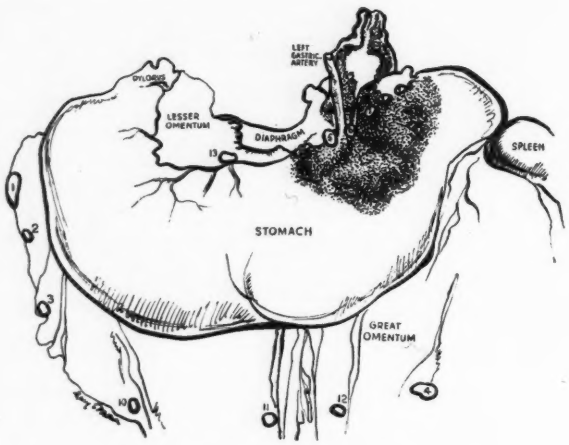


FIG. 21.—*Case* 4. Anterior aspect.

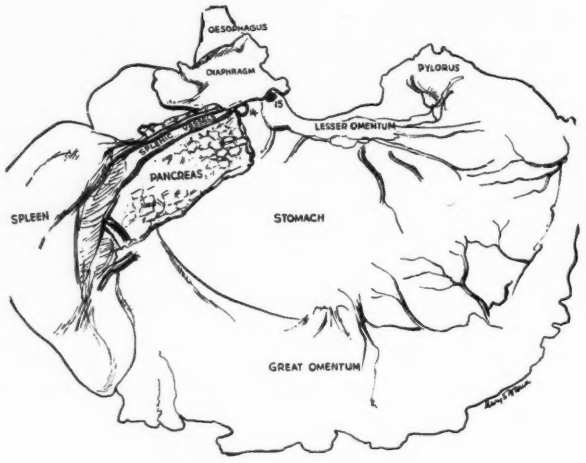


FIG. 22.—*Case* 4. Posterior aspect.

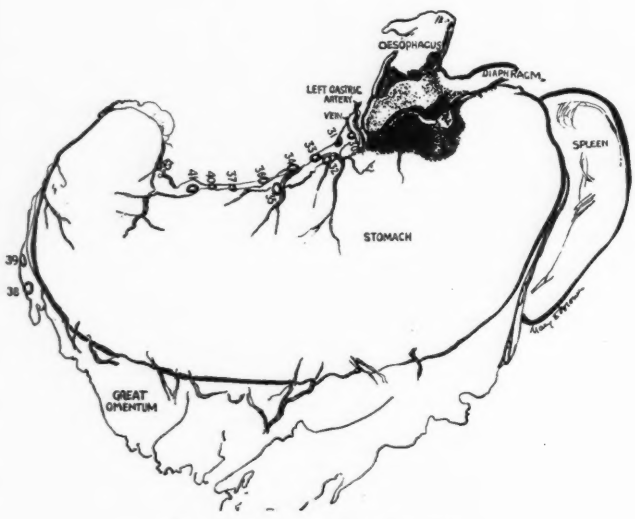


FIG. 23.—*Case* 5. Anterior aspect.

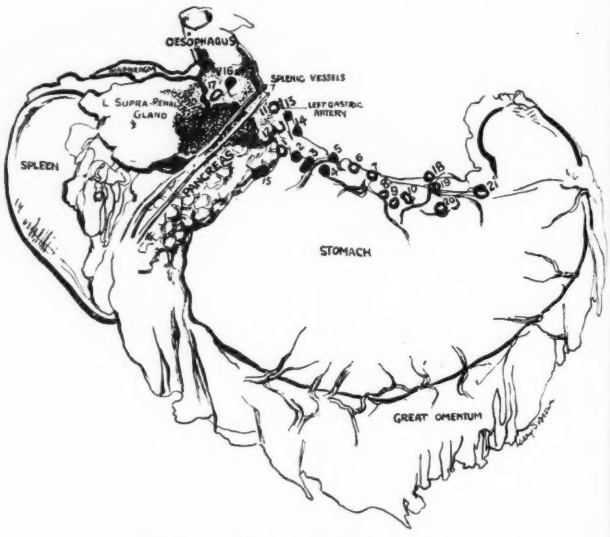


FIG. 24.—*Case* 5. Posterior aspect.

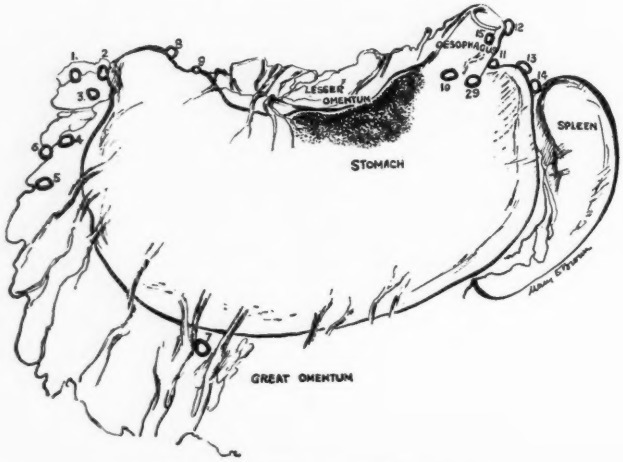


FIG. 25.—*Case* 6. Anterior aspect.

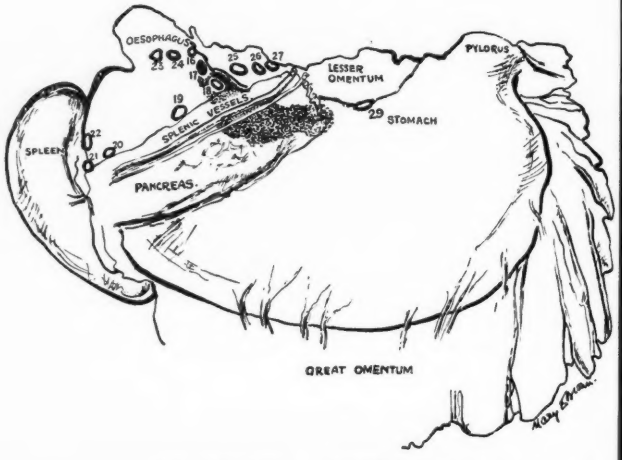


FIG. 26.—*Case* 6. Posterior aspect.

Case 4.—Mrs. E. B. (56), *carcinoma of stomach* (*Figs.* 21, 22).

Aug. 12, 1947: Total gastrectomy, splenectomy, omentectomy, heimi-pancreatectomy, and excision of lower third of œsophagus. Retrocolic Roux jejunal loop reconstruction.

Aug. 17: Died. Heart failure and broncho-pneumonia.

Pathology Summary: A well-differentiated adeno-carcinoma of the cardia invading the œsophagus and completely breaching the muscular wall of the stomach.

Metastases: In 1 gland. 14 dissected and examined.

Case 5.—W. L. (75), *carcinoma of stomach* (*Figs.* 23, 24).

Sept. 15, 1947: Total gastrectomy, omentectomy, splenectomy, hemi-pancreatectomy and partial œsopha-gectomy. Retrocolic Roux jejunal loop reconstruction. Left adrenalectomy.

Nov. 6: Died.

Pathology Summary: Adenocarcinoma of the cardia involving 3 cm. of the œsophagus. The cuff of growth in the stomach ranges from 0·5 cm. to 2 cm. in width. The left adrenal gland adherent and almost completely replaced by new growth.

Metastases: In 10 glands. 43 dissected. 42 exam-ined. Nodule No. 10 not a lymph-gland. Nodule No. 34 not sectioned as tissue had dried up.

Case 6.—J. D. (50), *carcinoma of stomach* (*Figs.* 25, 26).

Sept. 23, 1947: Total gastrectomy, splenectomy, omentectomy, hemi-pancreatectomy, and excision of lower third of œsophagus. Retrocolic Roux jejunal loop reconstruction.

Feb. 12, 1948: Is well.

Pathology Summary: Adenocarcinoma involving the lesser curvature of the stomach. Upper margin 1 cm. from the cardia. Lower margin 6 cm. from the pylorus. 12 cm. in diameter from above down, and 8 cm. across. Does not breach the muscle coat.

Metastasis: In unidentified tissue ? a Paccinian corpuscle. 29 glands examined.

Case 7.—A. H. (60), *carcinoma of stomach* (*Fig.* 27).

Aug. 25, 1947: Partial œsophago-gastrectomy to-gether with splenectomy, partial omentectomy, and pancreatectomy. Stomach used for reconstruction.

FIG. 27.—*Case* 7.

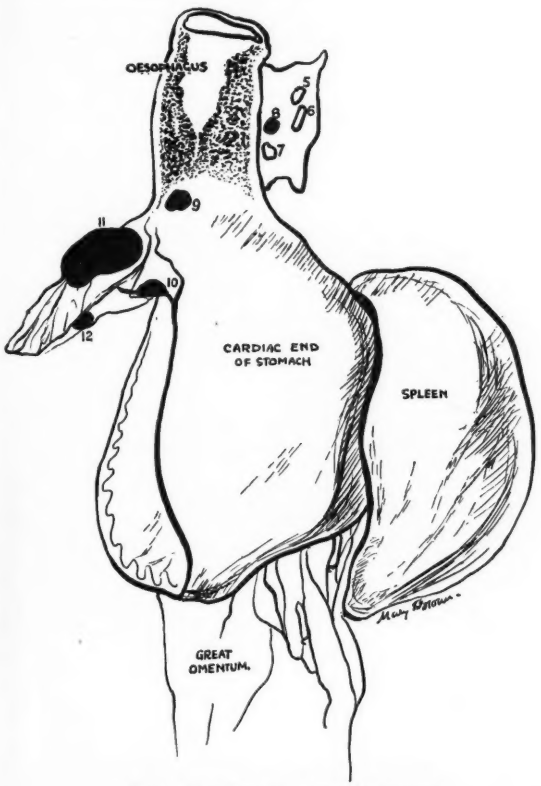


FIG. 28.—*Case* 8. Anterior aspect.

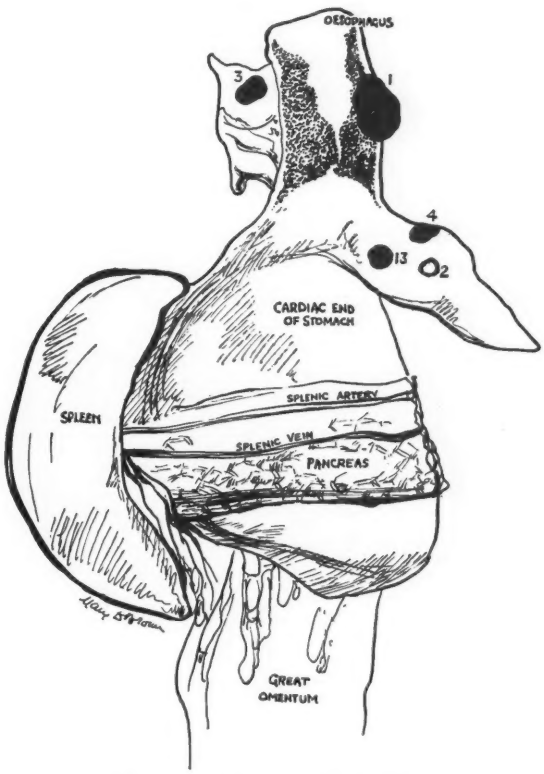


FIG. 29.—*Case* 8. Posterior aspect.

Jan. 2, 1948: Swallowing satisfactorily, but has post-operative peptic ulceration of the œsophagus above the anastomic line.

Feb. 19: Died.

Pathology Summary: Adenocarcinoma of the cardia which has extended up beneath the squamous epithelium of the œsophagus, and has penetrated the wall at the gastro-œsophageal junction, extending deeply into the cellular and adipose tissues around the cardia. There is perineural infiltration.

Metastases: In 2 lymph-glands. No. 2 and No. 6 invaded but not lymph-glands. No. 2, permeation of lymphatic vessels. No. 6, permeation of adipose tissue and nerves. 6 nodules examined.

Case 8.—Mrs. E. C. (56), *carcinoma of œsophagus* (*Figs.* 28, 29).

Oct. 30, 1947: Palliative partial œsophago-gastrectomy with splenectomy and partial pancreatectomy. Stomach used for reconstruction.

Nov. 25: Died from massive pulmonary embolus.

Pathology Summary: Anaplastic squamous-celled carcinoma. The lower end of the œsophagus is encircled by a mass of growth which involves about 4·5 cm. of its length and which extends into the stomach for 3 cm. Extensive invasion of cellular and adipose tissues around the cardia.

At the upper end the tumour extends beneath the unbroken squamous epithelium for a considerable distance.

Metastases: In 7 lymph-glands, and in two pieces of tissue (10, 13) not glands. 13 examined.

Case 9.—R. C. (54), *carcinoma of œsophagus* (*Figs.* 30, 31).

Nov. 1, 1947: Partial œsophago-gastrectomy. Splenectomy and partial pancreatectomy. Stomach used for reconstruction.

Dec. 12: Eating four small meals a day.

Pathology Summary: Squamous carcinoma completely and extensively breaching the muscular wall of the œsophagus. Extensive invasion up under normal squamous epithelium.

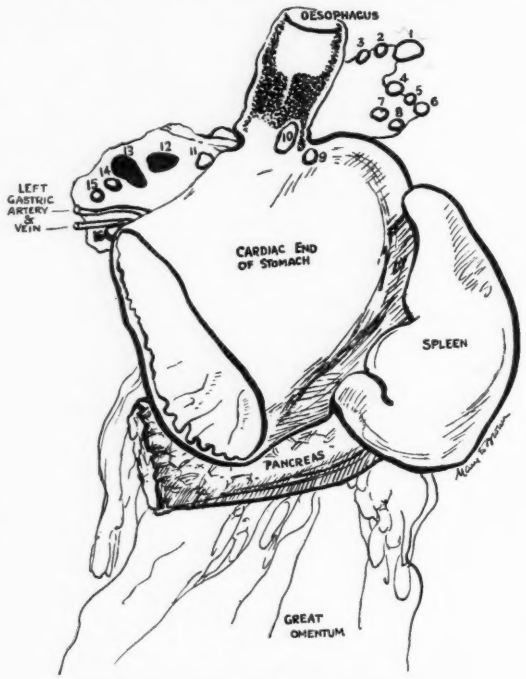


FIG. 30.—*Case* 9. Anterior aspect.

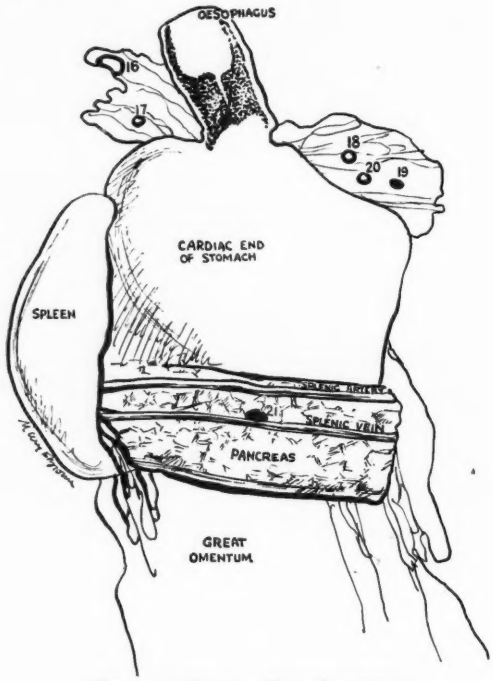


FIG. 31.—*Case* 9. Posterior aspect.

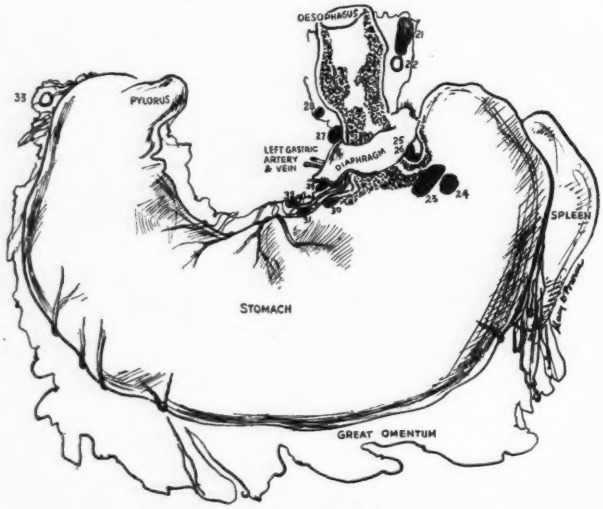


FIG. 32.—*Case* 10. Anterior aspect.

FIG. 33.—*Case* 10. Posterior aspect.

Metastases: In 2 lymph-glands and in 2 pieces of adipose tissue (19, 21) not identified as lymph-glands. 21 examined.

Case 10.—A. B. (65), *carcinoma of stomach* (*Figs.* 32, 33).

Nov. 15, 1947: Palliative total gastrectomy, splenectomy, omentectomy, partial œsophagectomy, and pancreatectomy. Roux jejunal loop reconstruction.

Jan. 16, 1948: Gained 1 st. in weight. Now 9 st.

April: Died.

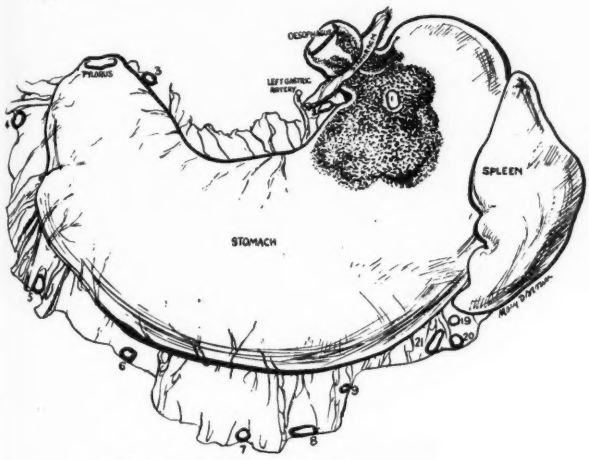


FIG. 34.—*Case* 11. Anterior aspect.

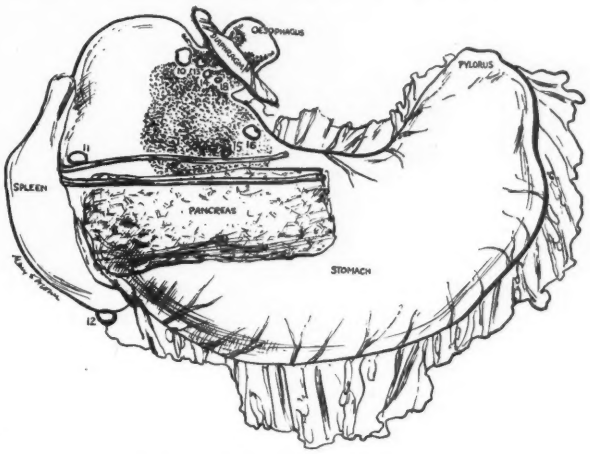


FIG. 35.—*Case* 11. Posterior aspect.

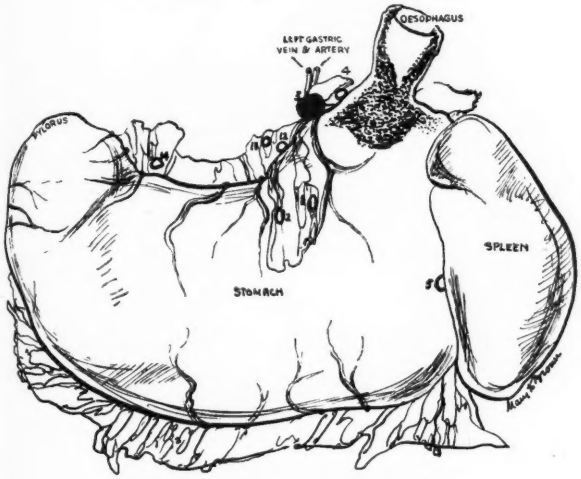


FIG. 36.—*Case* 12. Anterior aspect.

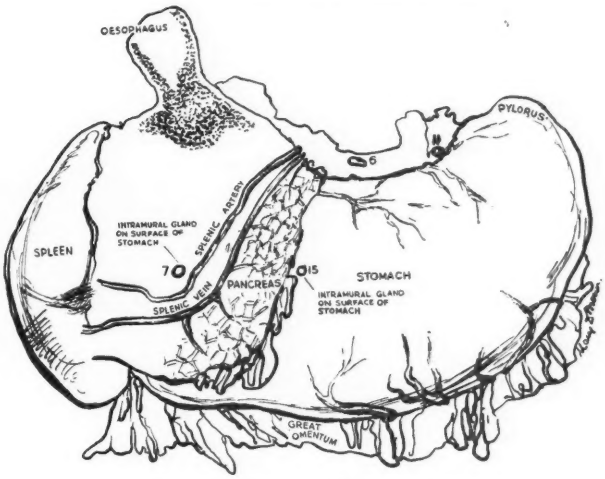


FIG. 37.—*Case* 12. Posterior aspect.

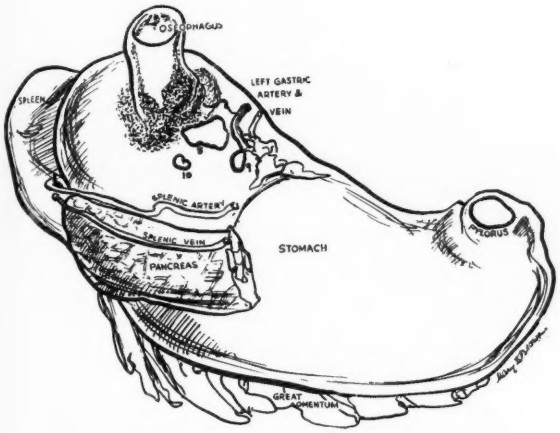


FIG. 38.—*Case* 12. Aerial view.

Pathology Summary: Anaplastic adenocarcinoma encircling the cardia and extending on to the posterior wall of the stomach for a distance of 2–3 cm. The growth appears to extend to the upper limit of removal of the œsophagus.

The gastric and œsophageal walls are completely breached by the tumour in the line of the lesser curvature of the stomach.

Metastases: In 23 glands. No. 26 and No. 28 invaded, but not lymph-glands. 33 nodules dissected. 31 examined. No. 11 and No. 15 were lost.

Case 11.—W. W. (61), *carcinoma of stomach* (*Figs.* 34, 35).

Dec. 4, 1947: Total gastrectomy, splenectomy, omentectomy, hemi-pancreatectomy, and partial œsophagectomy. Roux loop reconstruction.

Jan. 9, 1948: Discharged well.

Pathology Summary: Adenocarcinoma projecting as a rounded mass into the lumen of the stomach. The muscular wall of the stomach is completely breached and the perigastric cellular tissues are invaded. Much suppuration scattered throughout the growth.

No Metastases: 18 glands examined. No. 12 was a spleniculus. No. 17 (not charted), a vein. No. 19 not sectioned. Tissue had dried.

Case 12.—F. N. (69), *carcinoma of the œsophagus* (*Figs.* 36–38).

Jan. 1, 1948: Total gastrectomy, splenectomy, omentectomy and partial pancreatectomy, and œsophagectomy. Roux loop reconstruction.

Jan. 7: Died from post-operative bronchopneumonia.

Pathology Summary: A very cellular squamous carcinoma 2·4–4 cm. in length, encircling the lower end of the œsophagus. It has completely penetrated the wall of the gullet, and has stimulated a broad zone of dense fibrous reaction in front of its advancing edge.

The tumour has extended into and beneath the mucosa of the stomach for some distance. A little lower down in the stomach the tumour has erupted through the mucosa and presents on the surface.

Metastases: In 1 gland. 15 examined. No. 5 was not a gland. ? atheromatous artery. No. 11 was a *leiomyoma*, presumably of stomach wall.

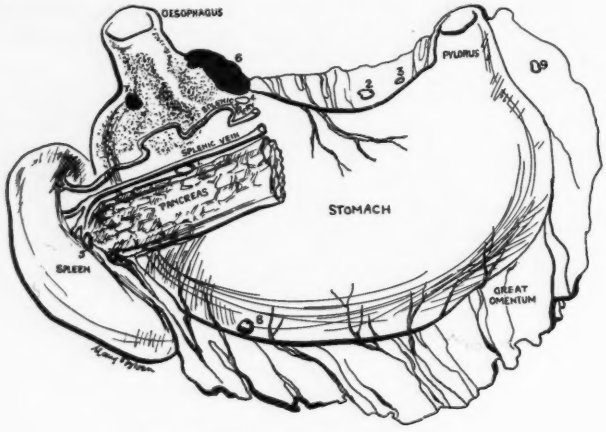


FIG. 39.—*Case* 13. Posterior aspect.

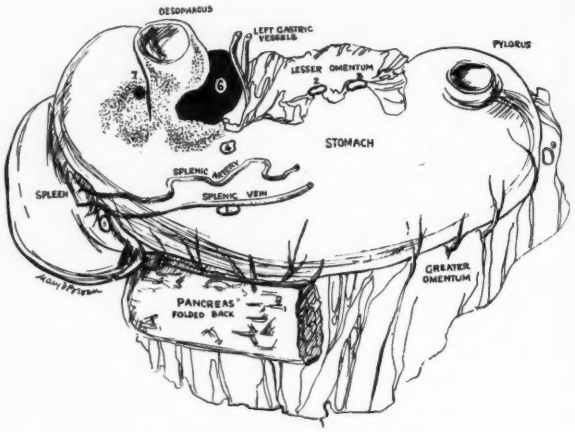


FIG. 40.—*Case* 13. Aerial view.

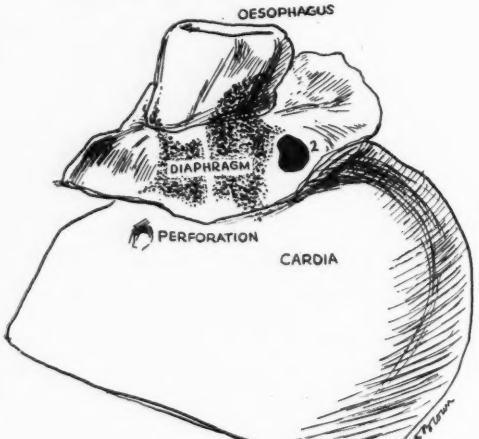


FIG. 41.—*Case* 14. Anterior aspect.

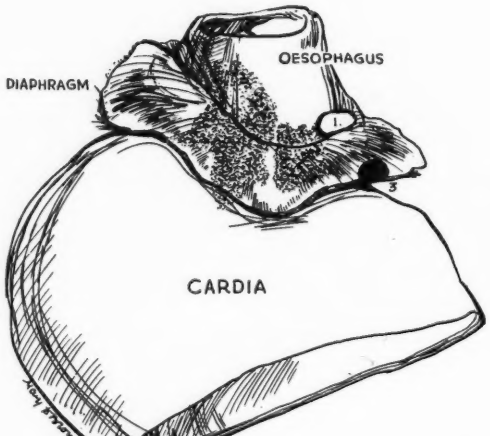


FIG. 42.—*Case* 14. Posterior aspect.

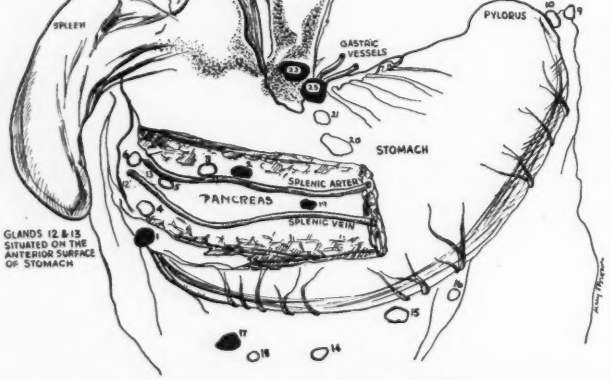


FIG. 43.—*Case* 15. Posterior aspect.

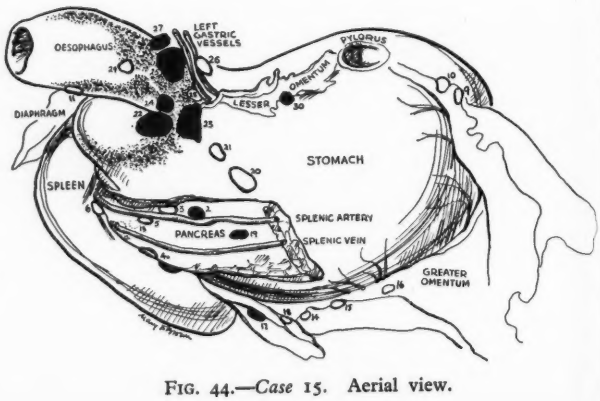


FIG. 44.—*Case* 15. Aerial view.

Case 13.—S. S. (64), *carcinoma of stomach* (*Figs.* 39, 40).

Jan. 29, 1948: Total gastrectomy and splenectomy, omentectomy, partial pancreatectomy, and partial œsophagectomy. Roux loop reconstruction.

Feb. 6: Patient died, following a leak at the anastomotic site, which developed on the sixth post-operative day.

Pathology Summary: A very cellular adenocarcinoma completely encircling the cardia. The growth extends more on the posterior wall of the stomach (for 5 cm.) than anteriorly, where only 1·5 cm. is involved. No tumour tissue has penetrated the pancreas, but there is a broad zone of dense fibrosis binding the tumour to the pancreas. Extensive sepsis is present at this site. At the upper limit of the growth there is deep undermining of the œsophageal mucosa. The muscular coat is completely breached.

Metastases: Present in 2 glands. 9 examined.

Case 14.—J. W. (82), *carcinoma of stomach* (*Figs.* 41, 42).

Jan. 21, 1948: Partial œsophago-gastrectomy together with splenectomy, partial omentectomy, and partial pancreatectomy. Stomach used for reconstruction.

Jan. 24: Patient died from post-operative bronchopneumonia.

Pathology Summary: Highly cellular adenocarcinoma 3 cm. in length. The growth has narrowed the œsophagus markedly, has completely breached the wall of the cardia, and has extended up beneath the squamous epithelium of the œsophagus.

Metastases: In 2 glands. 3 examined.

Case 15.—K. H. (51), *carcinoma of cardia* (*Figs.* 43, 44).

Feb. 17, 1948: Total gastrectomy, omentectomy, splenectomy, partial pancreatectomy, and partial œsophagectomy. Roux jejunal loop reconstruction.

March 16: Discharged. Swallowing well.

Pathology Summary: A very anaplastic and invasive tumour, partly glandular, partly spheroidal-celled, partly mucoid. It completely encircles the œsophagus, extends down into the stomach for 5 cm., and up the œsophagus for 2·5–3 cm. The muscular wall of the cardia is completely breached.

Metastases: In 10 glands and in 2 pieces of tissue (No. 28, 30) which are not glands. ? subperitoneal lymphatics. 30 nodules examined.

REFERENCES

JAMIESON, J. K., and DOBSON, J. F. (1907), *Lancet*, I, 1061.

LEIOMYOSARCOMA OF STOMACH

BY KENNETH WATSON

SURGEON, THE EAST SURREY HOSPITAL

INCIDENCE

BALFOUR and McCann (1930) report from the Mayo Clinic 4159 cases of malignant disease of the stomach, of which 54 were sarcoma, and, of these, 3 were leiomyosarcoma. Chaffin (1938) reviewed 363 cases of malignant smooth-muscle tumours of the stomach. D'Aunoy and Zoeller (1930), in a series of 100 cases of sarcoma of the stomach which were histologically proved, found that 28 were leiomyosarcoma. An incidence of approximately one in a thousand of malignant tumours of the stomach is accepted by most authors, although Schindler, Blomquist, Thompson, and Pettler (1946) suggest that better diagnosis would reveal a higher incidence than this. Holta (1943) suggests that a recent increase in the number of diagnoses of leiomyosarcoma has taken place at the expense of diagnoses of leiomyomas.

CASE REPORT

A. G. R., a male aged 63 years, was admitted to the medical wards on May 28, 1948, complaining of indigestion, dyspnœa on exertion, and frequency of micturition.

Past illnesses: Nil relevant.

Family history: Mother died of cancer aged 55. One sister died of heart disease aged 55. Details not known in either case.

Present illness: For some time prior to March, 1948, he had noticed that he was becoming more easily tired; he had also noticed increasing frequency of micturition during the past year. He did not regard either seriously and had not sought medical advice. On March 2 he had a sudden attack of pain under the left costal margin. The pain was related to respiration and was accompanied by a pyrexia of 99·6°. A provisional diagnosis of pleurisy was made. He was treated with sulphadiazine and the acute symptoms and pyrexia subsided within three days. He was kept in bed for three weeks, although a radiograph of the chest showed the lung fields clear. He had occasional cough with some "phlegm". On May 12 his doctor for the first time felt what was suspected of being an enlarged spleen. He was then seen by a consulting physician. On that occasion his main complaint was of windy dyspepsia. He also complained of feeling drowsy and had not got his accustomed energy. There was no complaint of pain, nausea, or vomiting. The spleen was noted as two fingerbreadths below the costal margin. There were some changes in the electrocardiogram of an inconclusive nature and one short period of auricular flutter during the examination. His blood-count showed Hb 60 per cent; R.B.C. 3,430,000; W.B.C. 23,000, of which polymorphs 73 per cent, eosinophils 2 per cent, lymphocytes 22 per cent, monocytes 3 per cent. E.S.R. 30 mm./hr. He was sent into hospital for investigation as a possible infective endocarditis.

ON ADMISSION.—A pale well-built man, not thin. Looks anæmic. C.V.S. apex beat 1 cm. outside midclavicular line. Loud apical systolic murmur present. Pulse 80, irregularly irregular but settled to a regular rhythm after a few minutes. B.P. 100/65. Temp. 99·9°. C.N.S. normal. Respiratory system, no abnormalities detected. Abdomen: spleen enlarged, colon loaded. Mouth, tongue, and fauces: Tongue, pale, smooth at edges. Teeth partly false, remainder good. P.R. prostate enlarged.

COURSE.—During the first two weeks in hospital he ran an evening pyrexia varying from 99·8° to 101·8°; during this period his morning temperatures were never above 98·6°. Blood-cultures were sterile. Urine was strictly normal and sterile on culture. An intravenous pyelogram showed some impairment in outline of the left kidney, a small diverticulum of the bladder, and much residual urine. The apparent size of the upper abdominal tumour was noted to vary very slightly; his heart was noted to be fibrillating on a number of occasions with intervals of regular rhythm. He had developed a harsh systolic murmur in the pulmonary area. Although no definite diagnosis was made he was given penicillin,

560,000 units daily in divided doses. After an initial drop to normal within 72 hours, the temperature commenced to swing again. A further examination of the blood showed R.B.C. 2,930,000; Hb 50 per cent; C.I. 0·9; W.B.C. 12,600, of which polymorphs 81 per cent; platelets 375,000.

June 19: I was asked to see the patient at this stage, largely for an opinion regarding his bladder-neck obstruction. I thought the mass in the left upper quadrant was not spleen alone but enlargement of the left lobe of the liver as well. I asked for further investigations to be done and the results were as follows: Blood-urea 37 mg.

FIG. 45.—Barium meal: The distal part of the stomach is filled with barium. The mucosal pattern of the proximal part is clear and the large cavity to the left of the greater curvature still filled with barium.

Fractional test-meal: "Curves for free and total acidity are within normal limits. No charcoal, bile, or blood present." Barium meal (*Fig.* 45): "(1) The stomach is displaced to the right and rather posteriorly. (2) The splenic flexure is depressed. (3) There is an irregular bilocular gas collection anterior and to the left of the greater curvature. It is not in the bowel and in the erect position contains a fluid level. (4) In the prone position barium freely enters and delineates both locules, some remaining therein after the stomach is empty. It does not enter large bowel from the cavity. (5) The spleen is not enlarged, diaphragm is of normal height and movement, no subphrenic gas present. (6) The stomach shows distortion due to pressure, no intrinsic lesion seen. Conclusion: This appears to be a gastric fistula with a subhepatic abscess on the left side. The fistula may be secondary to carcinoma of the stomach or a peptic ulcer or to an intrinsic lesion in the spleen, mesentery, pancreas, or liver. In view of its anterior position I think the spleen or liver more likely to be at fault as the primary cause of the abscess."

The patient's condition was now deteriorating he was fibrillating persistently and his anæmia worse, with R.B.C. 2,990,000; Hb 43 per cent; W.B.C. 14,500, polymorphs 78 per cent. With a history of an attack of sudden abdominal pain, persistent pyrexia, and a high leucocyte count I favoured the diagnosis of a subhepatic abscess due to a 'leaking' ulcer. He did not look ill enough to accept carcinoma of the stomach as the cause. The fibrillation was never fully controlled by digitalis. He was transferred to the surgical wards and given a pre-operative transfusion of two pints of blood very slowly.

FIRST OPERATION (July 25).—The morning of the day selected for operation he developed acute retention of urine. Anæsthetic: Pentothal–cyclopropane (Dr. S. deClive Lowe). A small left Kocher incision. Some pale yellow free fluid ran out on opening the peritoneum. The surface of a pale, hard, rubbery tumour was disclosed. The incision was enlarged sufficiently to get a hand in. The tumour was found to be attached to the greater curvature of the stomach by a broad pedicle; the remaining portion of stomach felt normal and could be invaginated into the mouth of an opening into the interior of the tumour. No enlarged glands or nodules felt in the liver. The tumour extended well back into the subhepatic area and had many adhesions, but gave the impression of being capable of being mobilized. A small piece of the surface was taken for biopsy and the wound closed. A suprapubic cystostomy was then performed and a de Pezzer catheter tied in. The tumour felt about the size of a large coconut. Following this operation he had three days of poor urinary output and pyrexia, but then improved. Great difficulty was experienced in controlling his fibrillation and some sepsis in the Kocher incision had to be dealt with. On the whole, however, his condition was rather better than expected. The biopsy report (Dr. L. P. B. Chamberlain) stated: "The section of tissue shows the presence of a degenerating cellular fibroma, there being no evidence of malignant disease". Multiple slow transfusions were now given, a total of three and a half pints of blood. The count after these showed R.B.C. 5,250,000, Hb 70 per cent. The urine was clear and ample. Wounds well healed.

SECOND OPERATION (Gastrectomy) (Aug. 25).—Anæsthetic: Pentothal, N_2O, trilene, curare (Dr. S. deClive Lowe). Saline drip started and stomach tube in situ. Upper left paramedian incision. The tumour appeared to have enlarged considerably since the first operation. There was a broad dense adhesion between the liver and the tumour anteriorly, many adhesions of a light character to the liver and spleen, and dense very vascular adhesions to omentum. The liver was separated first, the resulting surface was drawn together with mattress sutures and a light pad soaked in thrombin applied. The light adhesions were swept through with the hand and the adhesions to omentum clamped, divided, and ligatured. The tumour was now delivered, not without difficulty owing to sheer size. Hot packs were placed in the subhepatic space, where oozing was very free. The drip was changed from saline to blood. Clamps were applied to the stomach above and below the tumour and an extensive V resection performed, taking a good cuff of normal stomach wall. The packs and the thrombin pad on the liver were then removed; all bleeding was well contolled. The abdomen was then closed without drainage. Two pints of blood and one of saline were given during the operation. Following this operation he had the usual post-gastrectomy régime and improved rapidly. A striking feature was that the fibrillation became easily controllable and finally ceased altogether, even when digitalis was stopped—evidently purely toxic in origin. Three weeks after this operation he was up and about, feeling and looking well.

THE SPECIMEN.—The specimen weighed exactly 3 lb. and measured 20 × 15 × 9 cm. The section

report (Dr. L. P. B. Chamberlain) was as follows: "The microscopic examination of the tumour shows the presence of a leiomyosarcoma the cells of which show a fair amount of differentiation, isolated mitotic figures, and invasion of the stomach mucous membrane. There are areas of hyaline degeneration, necrosis, and hæmorrhages. Œdema is marked and there is a varying amount of fibrous stroma throughout the tumour".

FIG. 46.—View from in front showing tumour mass with cuff of normal stomach wall.

THIRD OPERATION (Second-stage retropubic prostatectomy—Millin's technique) (Sept. 22).—Anæsthetic: Heavy nupercaine—spinal. This presented no special feature or difficulty. The section of the prostate showed no evidence of malignancy. His post-operative course on this occasion was a little pyrexial but never occasioned anxiety. The catheter was removed in eight days; he passed urine per urethra without difficulty at once. The patient was retained in hospital until Oct. 12 in order to have a barium meal and a final opinion about his heart. Both reports were entirely satisfactory and he was allowed to proceed home.

PATHOLOGY

Microscopical.—A good description is that given by Lemon and Broders (1942): "Immature muscle cells of differing degrees of differentiation, grouped into bundles and bands which are separated by a fibrous stroma. Mitosis establishes malignant nature of the lesion." Platt, Walthemath, and Millhouse (1940) mention that the intercellular material is collagenous, as do many authors, but they and Schindler and others do not regard the presence of mitosis as absolutely necessary to establish the diagnosis. Baumgartner (1939) regards large spindles as the most common cell type.

Macroscopical.—A very rare intramural, infiltrating type, which simulates lymphosarcoma of the stomach is mentioned, but the great majority fall into two groups, endogastric and exogastric. These two groups have much in common pathologically, although clinically they behave differently. In

FIG. 47.—View looking into portion of stomach removed with tumour and showing opening into cavity in centre of tumour.

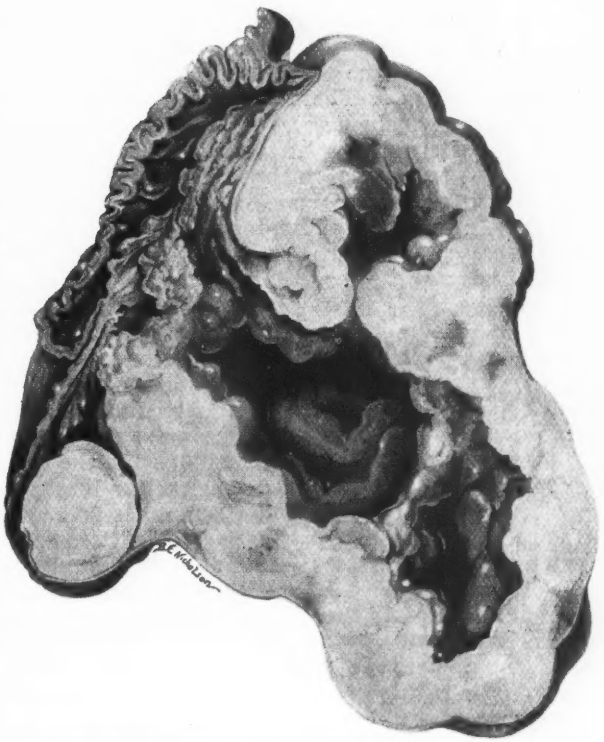


FIG. 48.—The specimen has been divided to show the size of the cavity in the tumour, the size of the communication with the stomach, and the apparent encapsulation.

both groups the tumours tend to rise from the curvatures (Ewing, 1928). Balfour and McCann (1930) state that they arise most commonly from the antrum, rarely from the pylorus, and never from the cardia.

Although Lattomus and Alden (1946) have recorded a massive case in this area, and Chaffin in his series recorded a few, this statement remains a fair summing up. The endogastric type range in size up to that of a grape-fruit and may be sessile or pedunculated. Central necrosis of the tumour is common, infection of the tumour accounting, in some cases, for the febrile state of the patient. The mucosa covering such tumours is almost normal except where central necrosis of the tumour has ulcerated through on to the surface. Infiltration of the stomach wall is slight in extent. Persistent bleeding from such tumours is a constant feature in the literature and in some cases mentioned by Bassler (1943) and Holta (1943) rapid severe bleeding has led to the early death of the patient. This does not occur with the exogastric type. The exogastric type are stated by Baumgartner (1939) to be the largest tumours of the stomach encountered in surgical work. Examples of such tumours weighing 18 lb. (McWhorter, 1919) and 13 lb. (Chaffin, 1938) occur in the literature. They are nodular and of a hard, rubbery consistency, but not as hard as carcinoma (Schindler and others, 1946). Central necrosis leading to a sinus communicating with the stomach is mentioned several times. Extensive adhesions to the liver and omentum are a common feature in the operation reports. There is frequently a limiting capsule present. Chronic hæmorrhage is common, but no instance of rapid bleeding is mentioned.

Malignancy.—Exceptionally a case may be found with a small tumour and widespread metastasis; such a one is described by Lyons and Schneider (1943). The great majority show a very low degree of malignancy. Holta gives the average duration of symptoms before seeking treatment as 3½ years, a statement which agrees exactly with the experience of Lemon and Broders in their series (1942). The latter authors suggest metastasis as being present in from one-third to three-quarters of all cases; this of course includes cases diagnosed at necropsy. The reports available show a very small incidence of metastasis discovered at operation but recurrence locally in the liver within 2 to 4 years is frequent; on the other hand survival for as long as 14 years after resection has been recorded.

One may say that a small minority of the cases show a short and explosive course but the great majority have a long history of symptoms preceding the seeking of medical advice. In this latter group extensive local disease is found with clinical and histological evidence of malignancy, but not as a rule metastasis, and that metastasis following removal is uncertain or very slow. One is forced to suspect that the majority of these growths were simple tumours initially, that the onset of malignancy was accompanied by a phase of marked local change but not by rapid metastasis, and that the small group with short history and rapid course were sarcoma *ab initio*.

CLINICAL FEATURES

Anæmia is the one feature common to all cases. This is usually severe—Lemon and Broders give an average Hb 46 per cent for their series; this is a fair reflection of other reports, and the condition has been mistaken for pernicious anæmia. Following this, in order of frequency, come: (1) pain, epigastric or left upper quadrant, sometimes severe; (2) a palpable tumour—this occurs in over 40 per cent of recorded cases. Other features which may occur, but not with any regularity, are hæmaturia or melæna and pyrexia due to infection of the tumour mass. Negative observations of value are that neither free nor total acidity is affected and that loss of weight has been small or even negligible. Radiography will usually confirm the existence of a gastric lesion if a careful technique is used; the correct diagnosis, however, was only once put forward in all the cases reviewed. Gastroscopy has proved useful in confirming the existence of a gastric lesion, but even Schindler with his immense experience remarks that in none of his cases was a definite diagnosis made. In general it may be stated that there is no characteristic clinical feature, that the endogastric tumours call attention to themselves earlier than the exogastric, mainly due to earlier and more severe secondary anæmia; and that provided it is recognized that the stomach is the cause of the patient's illness the majority of the cases will lead to the appropriate treatment being instituted.

DIFFERENTIAL DIAGNOSIS

It is clear that an enumeration of all the possible diagnoses would be wearisome. Assuming that the existence of a tumour of the stomach is accepted, two conditions will need more detailed consideration: (1) Carcinoma—the most striking point is that the course of a carcinoma of the stomach is far more rapid. A patient with a palpable mass due to carcinoma will as a rule be far more cachectic than these cases, and the mass, if due to carcinoma, will usually be fixed whereas if due to sarcoma this is not so. (2) Lymphosarcoma—this is a purely infiltrative lesion and if encountered at operation should not be subjected to resection. A biopsy should, however, be taken, as it is distinctly radiosensitive and radiation is the correct treatment.

TREATMENT

This is purely surgical. Platt, Walthemath, and Millhouse (1940) state that these tumours are radioresistant and Schindler mentions a case which was explored before and after radiotherapy: no effect whatever had been produced by the radiation. The point to remember is that these cases, particularly the exogastric variety, may easily appear inoperable. The large size of the tumour and the poor condition of the patient do not tempt one to embark on operative surgery. With the abdomen open the apparent magnitude of the task, the vascularity, and the extent of the adhesions, appear to confirm these impressions. Nevertheless, the slow rate of metastasis, the slight degree of infiltration, and the knowledge that you may be dealing with this type of tumour should urge one on. Chaffin (1938) points

out that the results of conservative excision are as good as those of more radical resection, with a lower mortality. He mentions an operative mortality of 8 per cent and a further 5 per cent in the post-operative period. Schindler and others, in their final comment, remark: "In leiomyosarcoma the surgeon cannot easily be too courageous."

SUMMARY

1. A résumé of the clinical and pathological features of leiomyosarcoma of the stomach.

2. A case of exogastric leiomyosarcoma exhibiting typical features.

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THE DIAGNOSIS AND TREATMENT OF VASCULAR DISEASES*

WITH SPECIAL CONSIDERATION OF CLINICAL PLETHYSMOGRAPHY AND THE SURGICAL PHYSIOLOGY OF THE AUTONOMIC NERVOUS SYSTEM

BY PROFESSOR R. H. GOETZ

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MR. PRESIDENT, MEMBERS OF THE COUNCIL, LADIES AND GENTLEMEN, I find it difficult to express adequately my appreciation of the great honour you have bestowed on me and the University of Cape Town in electing me to deliver this lecture.

I am sure you will forgive me, that in my thanks to you I also record my gratitude to two of my former teachers whose generosity and guidance in the early days made this honour possible for me.

I refer to Professor I. de Burgh Daly, now in Cambridge, and to Professor Charles F. M. Saint in Cape Town, who took me under their wings when I first became a member of this British Commonwealth of Nations.

I am indeed sensible of the great men who graced this platform before me. It is little wonder that I am filled with a sense of utter humbleness.

INTRODUCTION AND CLASSIFICATION OF PERIPHERAL (ARTERIAL) VASCULAR DISEASES (P.V.D.)

There has been a marked change within recent years in the attitude of the medical profession towards patients afflicted with vascular disease. It is realized that with modern methods of treatment not every case need be mutilated by amputations, and the study of peripheral vascular diseases (P.V.D.) is rapidly being accepted as a new branch in medical science. Despite this, many medical schools still provide inadequate training for its study and care, and practitioners only too frequently fail to recognize its manifestations until gangrene has occurred.

Although in the past their potential significance had been greatly underestimated, to-day it is fully realized that the development of P.V.D., particularly if we include, as we must, hypertension and arteriosclerosis, constitutes one of the vastest of all medical problems for the future. Peripheral vascular disease is, no doubt, more frequently recognized as such, and as the cause of not only peripheral but widespread systemic pathology.

We are familiar with arteriosclerosis being a generalized condition, but thrombo-angiitis obliterans, Raynaud's phenomenon, scleroderma, and many other so-called peripheral vascular diseases are clearly not confined to the limbs only, but affect the vessels in other organs as well, such as the heart, the brain, the lungs, the intestine, and the kidneys. On the other hand, there are many systemic diseases, such as periarteritis, dermatomyositis, diabetes, lupus erythematosus, and Boek's sarcoidosis, which may present with vascular signs and symptoms in the extremities. Although this presentation deals only with the circulation in the extremities, the term 'peripheral vascular disease' should not be confined to them only, but should be broadened to include diseases of any blood-vessel outside the heart.

Unfortunately there does not exist a completely satisfactory classification, and in fact some authors have classified vascular diseases alphabetically. Even the most recent text-books omit a classification altogether. There can be no doubt that a reasonably good classification would be of great help to the practitioner and would greatly assist in diagnosis. I have therefore attempted to sort out the conditions and propose the following:—

* Being the first part of an Arris and Gale Lecture delivered before the Royal College of Surgeons of England on Jan. 13 and 14, 1949. The second part will appear in the October number.

CLASSIFICATION OF PERIPHERAL (ARTERIAL) VASCULAR DISEASES

I. Non-organic (Dynamic or Functional) Vascular Disorders.—

A. LOCAL.—
1. *Vasoconstrictor.*—
a. Primary (hereditary).—
Cold extremities.
Acrocyanosis
Raynaud's ' disease ' (primary or idiopathic)
Erythrocyanosis
Levido reticularis
b. Secondary.—
Traumatic.—
Arterial stupor
Minor causalgias following blows, thrombophlebitis, fractures
Spasm associating embolism
Exposure to cold.—
Frost-bite
Pernio
Secondary Raynaud's phenomenon due to mechanical irritation.—
Cervical rib or its equivalents
Scalenus anticus syndrome
Pneumatic tools
Crutches
As initial symptom of organic vascular disease.
Toxic vasospasm.—
Ergot poisoning
Arsenic
Nicotine
Neurotrophic disorders.—
Spina bifida
Poliomyelitis
Hemiplegias
Tumours and lesions to brain and spinal cord
Syringomyelia
Others.—
Autohæmo-agglutination
2. *Vasodilator.*—
Erythromelalgia (erythermalgia).—
Primary
Secondary: in arteriosclerosis, polycythæmia, trauma
Erythralgia
Post-traumatic bone atrophy (Sudeck's)
Vasodilation associating arteriovenous fistula
Immersion foot (hyperæmic stage)

B. GENERAL.—
1. *Vasoconstrictor.*—
Essential hypertension
Endocrine disorders, e.g., menopausal, hypothyroidism
2. *Vasodilator.*—
Hyperthyroidism
Vitamin-B deficiency (chronic alcoholism)

II. Organic (Structural) Vascular Diseases.—
Spastic element additional in many conditions.

A. CONGENITAL.—
1. *Coarctation of aorta*
2. *Arteriovenous fistula (congenital)*
3. *Other congenital anomalies*

B. NON-INFLAMMATORY AND DEGENERATIVE LESIONS.—
1. *Local.*—
Acute vascular accidents.—
Emboli of various types
Traumatic occlusion
Ligation, severance
Arteriovenous fistula (acquired)
Bilateral gangrene (so-called Raynaud's gangrene)
Gangrene due to escharotics, e.g., carbolic acid.
Ayerza's syndrome (sclerosis of pulmonary arteries)
2. *General.*—
Arteriosclerosis and atheromatosis
Diabetes
Occlusions relative to disorders of blood.—
Polycythæmia vera
Essential thrombophilia secondary to hypertension

C. INFLAMMATORY.—
1. *Acute.*—Arteritis secondary to acute infectious diseases, e.g., rheumatic arteritis
Effects of environmental temperature on vascular system.—
Pernio
Immersion foot
2. *Subacute.*—
Periarteritis nodosa
Luetic arteritis
Tuberculous arteritis
Ergot
3. *Chronic.*—
Thrombo-angiitis obliterans (Buerger's disease)
Chronic toxic arteritis
Cervical rib causing chronic inflammation of the arterial wall
Aneurysms

D. NEOPLASTIC CONDITIONS.—
1. *Hæmangiomas*
2. *Glomus tumours—hæmangiopericytomas*
3. *Hæmangio-endotheliomas*
4. *Angiosarcomas*

There are two main groups—namely, non-organic, or functional, vascular *disorders*, and organic vascular *diseases*. The vascular disorders may be subdivided into vasoconstrictor and vasodilator disorders, the organic diseases conventionally into congenital, non-inflammatory, inflammatory, and neoplastic conditions.

The difficulty arises in the fact that we are not dealing with water-tight compartments, and that there are mixed cases. Many cases start as functional disorders and go on to organic arterial disease. It is the object of special tests to measure the circulation in order to determine whether we are dealing with the functional or organic type and to assess the degree of each in a mixed case.

The methods generally used for measuring the peripheral blood-flow in man, like skin temperature and oscillometric readings, furnish, however, indirect indices of blood-flow only or they do not deal with the peripheral circulation in the strict sense. For

quantitative measurements plethysmography has to be resorted to.

No doubt you are all familiar with the various types of limb plethysmographs which have furnished us with extremely valuable information concerning the regulation of the peripheral circulation. I need only mention the brilliant work of Sir Thomas Lewis and his school. However, these apparatuses are cumbersome, difficult to manage, and can hardly be used in the wards or in the operating theatre. For this reason most clinicians have a prejudice against plethysmography and regard it still to-day as a laboratory method not suitable for routine clinical investigations.

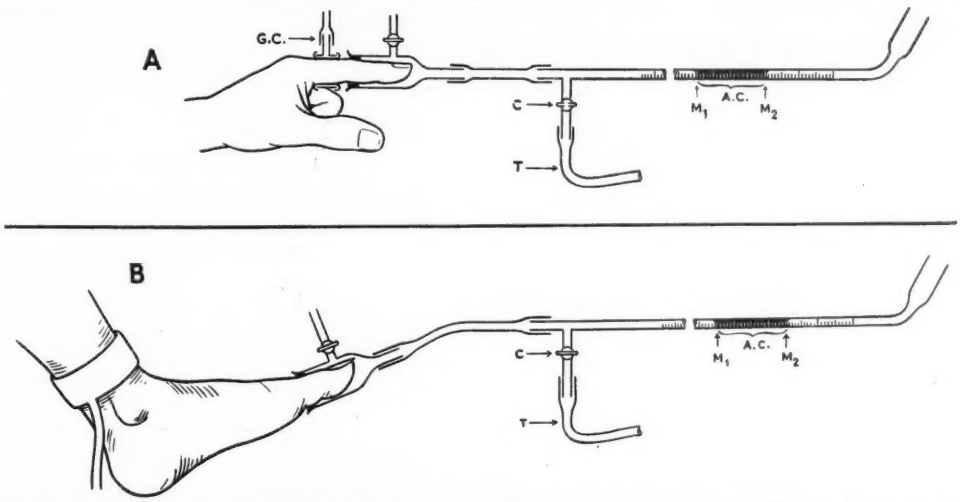

FIG. 49.—The principle of the optical (digital) plethysmograph, (A) as applied for the finger, (B) as applied for the toe. A.C., Alcohol column in the pipette; C, Tap; T, Rubber tubing for adjusting alcohol column; M_1 and M_2, Menisci of the alcohol column, the movements of which are recorded; G.C., Gaertner's cuff for applying venous congestion during the venous congestion test.

It is the purpose of this lecture to demonstrate to you our portable digital plethysmograph which is simple in its application, lends itself easily to routine clinical investigations in the wards, the theatre, or in the patient's home, and which furnishes us with accurate information concerning the peripheral blood-flow which is difficult to obtain by any other method. The results obtained are not only strictly referable to the blood-flow through the skin but they are also representative of the peripheral, the *ultimate*, circulation, in contrast to the oscillometric readings, which refer to what we have termed the '*penultimate*' circulation.*

I am fully aware that in stressing the importance of measuring the peripheral blood-flow, the blood-flow through the skin, I will be accused by many workers of underestimating the value of such methods which register the blood-flow through muscles, and of minimizing the importance of symptoms and signs arising from a deficiency of the blood-flow through the muscles. Nothing would be less true. Indeed, intermittent claudication is one of the most valuable symptoms, and we should question every case most carefully in this respect. However, the danger to the limb, and often to the life of the patient, does not come from the intermittent claudication. I cannot recall a single case where we were forced to amputate the limb for intermittent claudication. The danger comes from a deficient circulation of the skin, from threatening or developed gangrene. It is for this reason that assessment of the blood-flow through the skin is of such importance. It is therefore essential that we employ accurate scientific methods of measuring the peripheral blood-flow in every patient, enabling us to form a proper picture of the state of the peripheral circulation at any one moment.

THE PRINCIPLE OF THE METHOD

The changes in the peripheral blood-flow are continuously recorded by enclosing a digit (finger or toe) in a suitable glass container (plethysmograph proper), which is connected to a specially constructed pipette graduated in one-hundreds of a c.c. and containing a column of alcohol (*Fig.* 49). Great care is taken to avoid any interference with the circulation by using too tight a plethysmograph. The plethysmograph is sealed with soft surgical petroleum jelly in order not to interfere with the circulation as does occur with other methods using rubber diaphragms, putty, or plaster-of-Paris. Any change in the volume of the digit will then be transmitted to the alcohol column of the pipette, the meniscus of which is enlarged and projected (*Fig.* 50) on the photographic paper of a recording camera. The pipette is mounted horizontally and fitted with a T-piece and tap permitting adjustment of the alcohol column during the experiment (*Fig.* 51). Therefore changes in volume are not accompanied by changes in pressure within the system, and no interference with the capillary circulation occurs, measurement being under most physiological conditions. Until recently we have only recorded the blood-flow through one digit at a time, but in this instrument two pipettes are mounted above each other and the blood-flow from two limbs can therefore simultaneously be recorded (*Fig.* 51). The advantage of this is obvious. It permits comparison of blood-flow through the upper

* It is with gratitude that I put on record my indebtedness to Mr. B. Shub, Cape Town, without whose financial help it would have been impossible to develop this instrument.

and lower extremities, through a normal and sympathectomized limb, or through a normal and a diseased limb, or any other combination.

Simultaneously with the plethysmogram the following can be recorded: The respiratory movements, the skin temperatures by means of thermocouples (a thermos flask for the reference junction has been built in), and the pressure in the cuffs

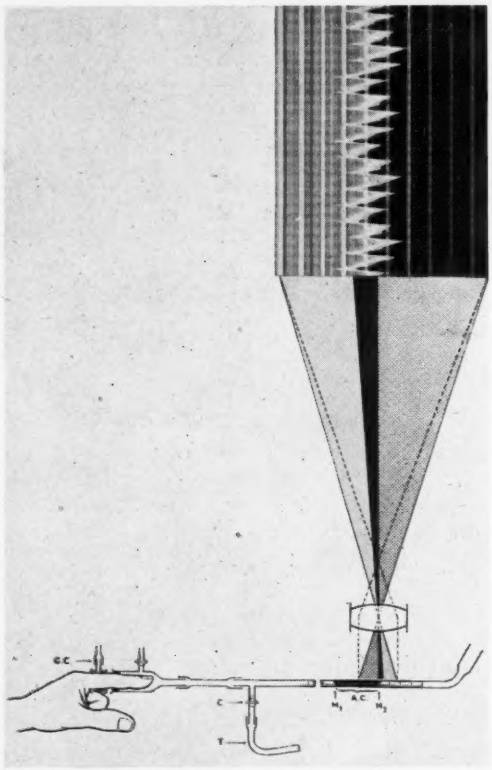

FIG. 50.—The optical principle underlying the recording of the movements of the meniscus. The pipette filled with alcohol acts like a biconvex lens and intensifies the light, giving the appearance of dark grey or black on the film. The pipette containing the air behaves to the light like two plane-parallel plates. The light, therefore, just passes through it, giving the light-grey shade on the film. The meniscus, being curved, reflects the light and casts a shadow on the film, hence the white line between the dark and light shades of grey. The calibration on the pipette absorbs the light and therefore appears as white lines on the film. The system is therefore always calibrated whatever the enlargement. For abbreviations, *see Fig.* 49.

during venous congestion (*see* p. 36) while measuring the arterial inflow (venous occlusion plethysmography). The capsules provided for the recording of the respiration and the occlusion pressures may alternatively be employed for recording arterial or any other pressures simultaneously with the peripheral blood-flow. The galvanometer, on the other hand, may be used for recording skin resistances or the current from a photo-electric cell. The apparatus is therefore a universal instrument for the study of the peripheral circulation both in health and disease.*

The camera has the following features, amongst others: daylight loading, built-in timing device, six speeds (2/25 mm./second), 150-ft. magazine, 120-mm. standard recording paper, and may be used independently for any other method when photographic recording is required.

Although the investigations can easily be carried out with the patient in bed, either in the ward or at his home, in the operating theatre or the laboratory, it is preferable to investigate all patients under identical conditions. In Cape Town we therefore use a specially constructed couch (Goetz, 1948, a), and if possible the routine examinations are conducted in a draught-free and noiseless room the temperature of which is being kept constant at 20° C.

THE EVALUATION OF THE PLETHYSMOGRAM

Our plethysmographic method is sensitive enough to register in all its detail the volume changes occurring with each heart-beat (the pulse-volume) besides recording the changes in the volume of the digit as a whole (digital volume) (*Fig.* 52). The pulse-volume (*Fig.* 52 A) represents the difference between the arterial inflow and the venous return during a single heart-beat, while the digital volume (*Fig.* 52 C) reflects mainly changes in the filling of the cutaneous capillary and venous plexus, which take place over many cardiac and one or more respiratory cycles.

In the normal the height of the pulse-volume depends mainly on three factors: the heart output per beat, the peripheral resistance (vasomotor tone of the arterioles), and the venous return. We may assume that during the rest the heart output and venous return will remain constant during the course of the test. The pulse-volume, therefore, depends mainly upon the peripheral resistance and indicates the state of the smallest arteries and arterioles. It is larger when the arteries and arterioles are relaxed, as after local or reflex heating, but is small if the vessels are constricted, as after the application of cold or the injection of vasoconstrictor substances.

Normally the vessels are neither fully dilated nor fully constricted, but are steering a middle course determined by the vasomotor centre according to the needs of body temperature regulation. Indeed, the height of the pulse-volume is an exquisite indicator of such sympathetic vasomotor activity as is of insufficient degree to show itself in blood-pressure changes. Even the slightest change in the central vasomotor tone will be reflected in changes of the height of the pulse-volume (*Fig.* 52 C). There occurs a decrease in pulse-volume with any untoward stimulus such as pain, excitement, expectancy of pain, unpleasant auditory stimuli, a deep breath, a yawn, and any similar stimulus (*Fig.* 53). A small pulse-volume during rest may therefore be due to structural changes or to a high vasomotor tone. It is therefore insufficient for clinical purposes to take recordings of the blood-flow at random or during rest only. In each case the vessels have first to be relaxed to full vasodilatation, and only then are we in a position to judge whether the vessels are normal or whether the diminution in blood-flow is organic in origin (*Fig.* 54).

Although many methods have been devised to achieve vasodilatation or ablation of sympathetic tone, such as spinal anæsthesia, typhoid vaccine injections,

* The apparatus will be obtainable from Hilger and Watts Ltd., Hilger Division, 98, St. Pancras Way, London, N.W.1.

peripheral nerve and paravertebral blocks, immersion of one extremity in a tank of water of 45° C. for 30 minutes and covering the subject with blankets to prevent the dissipation of heat is still the simplest and least objectionable method of producing paralysis of central vasomotor tone. It can be repeated *ad lib*, and vasodilatation once initiated is rapid and complete.

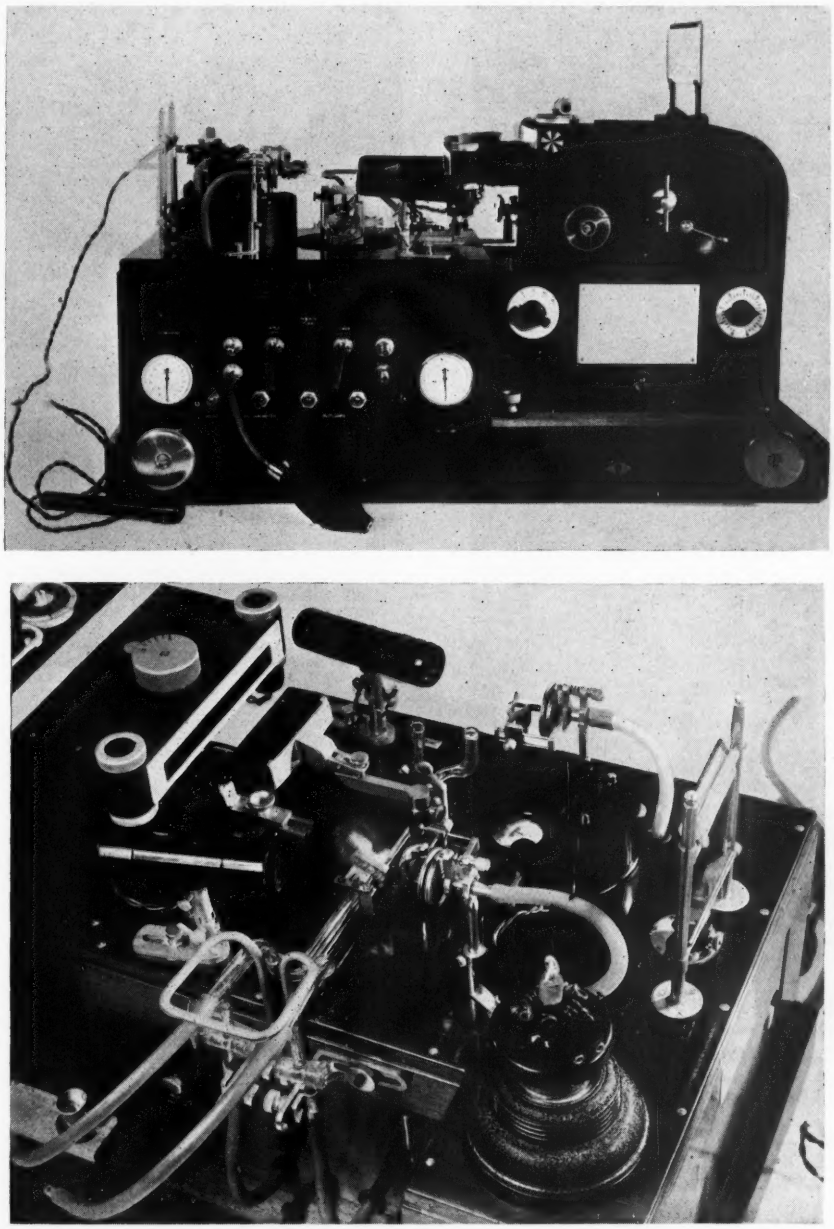

FIG. 51.—Photographs of the portable plethysmograph. Note the two pipettes mounted horizontally, the galvanometer for the recording of the skin temperature or skin resistance, the capsules for recording respiration or pressures, and the built-in reservoir with manometer for carrying out venous congestion tests recording the pressure simultaneously.

In *Fig.* 55 the effect of body heating on the height of the pulse-volume is well illustrated. Before body heating (during rest) the pulse-volume recorded only 0·002 c.c. After 15 minutes' body heating it has risen to 0·01 c.c., and with vessels fully dilated the pulse-volume reached 0·025 c.c.

Naturally vasodilatation affects not only the arterioles but all vessels, and in particular the capillary and the venous plexus in the skin, the filling of which is reflected by a marked increase in the digital volume. By taking a continuous record of the plethysmogram during body heating, this increase in the digital volume can be measured and amounts to about 5 per cent of the digital volume enclosed (*Fig.* 55). It is important to realize that in purely arterial disease body heating will demonstrate a diminution of the pulse-volume during full vasodilatation while there is still the normal response in the digital volume. There are other conditions where the reverse holds good, the digital volume

being affected to a greater degree than the pulse-volume, such as scleroderma and œdema. It appears from experiments reported elsewhere that the behaviour of the subpapillary venous plexus may play a greater role in skin temperature regulation than has hitherto been suspected (Goetz, 1949).

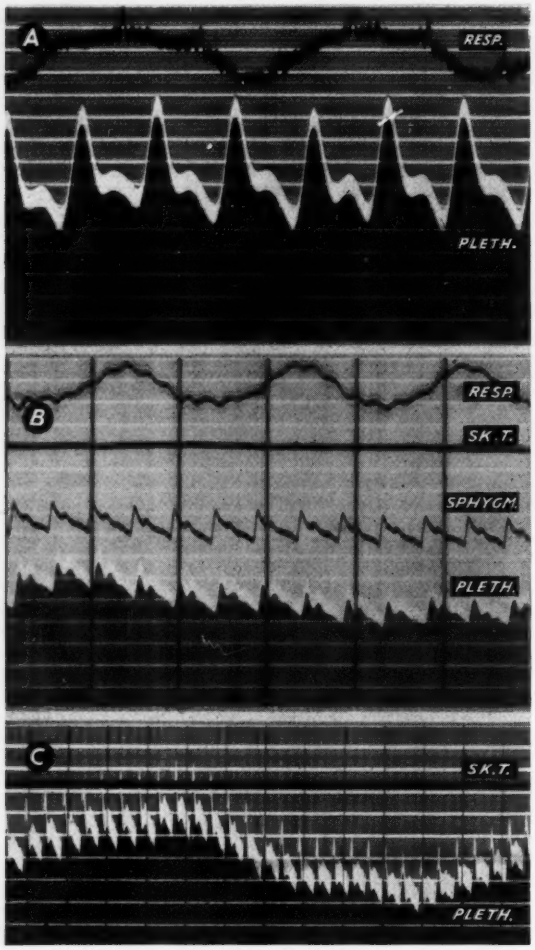

FIG. 52.—A, Plethysmogram (first right toe) and respiration simultaneously recorded at fast speed to demonstrate the shape of the individual pulse-volume. B, Pulse-volume (third right finger), sphygmogram (radial artery), skin temperature, and respiration all simultaneously recorded in a normal subject moderately dilated. (Room temperature 20° C.) C, Plethysmogram (second left finger) taken at the standard speed (5 mm. per second) to demonstrate spontaneous fluctuations in pulse-volume and digital volume. Skin temperature simultaneously recorded. In all tracings, unless otherwise stated, the white horizontal calibration lines indicate changes in volume of 0·01 c.c. and changes in skin temperatures of 0·2° C. Ordinates, 2 seconds.

In *Fig.* 55 a chart has been prepared of the effect of body heating on the peripheral blood-flow in the normal, as obtained by the continuous and simultaneous recording of the plethysmogram and the skin temperature. As can be seen, the pulse-volume is the first to respond to the relaxation of the vasomotor tone, followed by the digital volume. There is a definite lag in the response of the skin temperature which is easily explained by the fact that the skin temperature depends to a great extent upon the filling of the capillary and venous plexus. Dilatation once initiated is rapid, and after twenty minutes the pulse-volume usually reaches 0·02 c.c. or more and

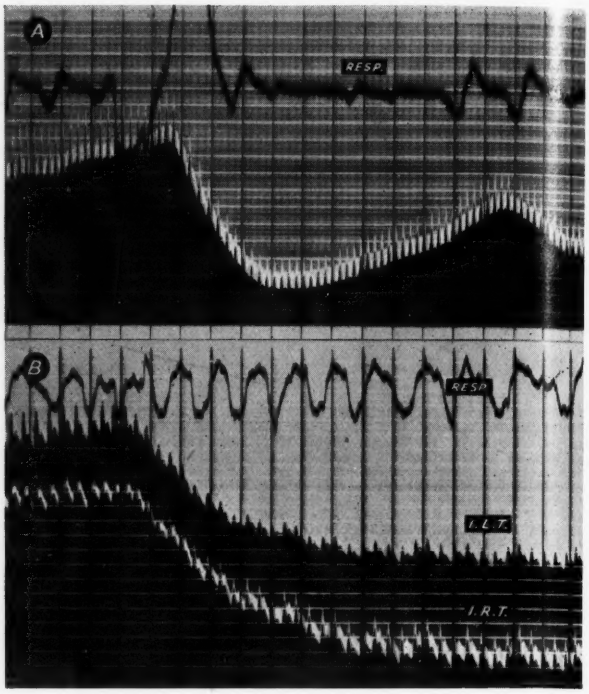

FIG. 53.—A, Plethysmogram (third left finger) and respiration simultaneously recorded to demonstrate reflex vasoconstriction resulting from an accidental deep breath. B, The effect of calculating 19 × 16 on the blood-flow in the lower limbs. Blood-flow in both big toes simultaneously recorded. Note decrease in the height of the pulse-volume and the digital volume.

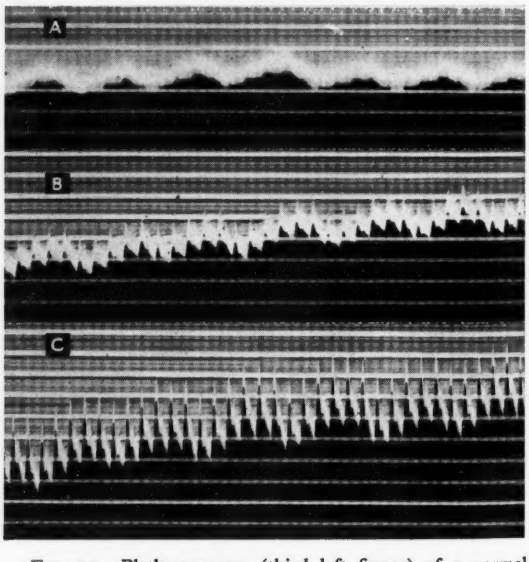

FIG. 54.—Plethysmogram (third left finger) of a normal subject, demonstrating the relation between the height of the pulse-volume and the degree of vasomotor tone. A, Fully constricted; B, During dilatation; C, Fully dilated.

the digital volume is reaching its peak. The skin temperature follows, to reach values between 33° and 34° C. If the subject has been fully constricted at the outset, there will be a delay in the onset of the vasodilatation, and cases of Raynaud's phenomenon may take easily up to ten minutes before starting to dilate.

In the normal the height of the pulse-volume varies between 0·002 c.c. during maximum constriction and 0·025 c.c. during full vasodilatation. Higher values are met with, particularly in adolescence and in such conditions as acrocyanosis. From examining a large number of normals we have come to regard a pulse-volume of 0·02 c.c. during full vasodilatation as the 'minimum vasodilatation level'. If the pulse-volume during full vasodilatation reaches this level then we are confident that there is no arterial disease. However, any value below 0·02 c.c. has to be accounted for, and is usually due to structural changes in the arterial tree.*

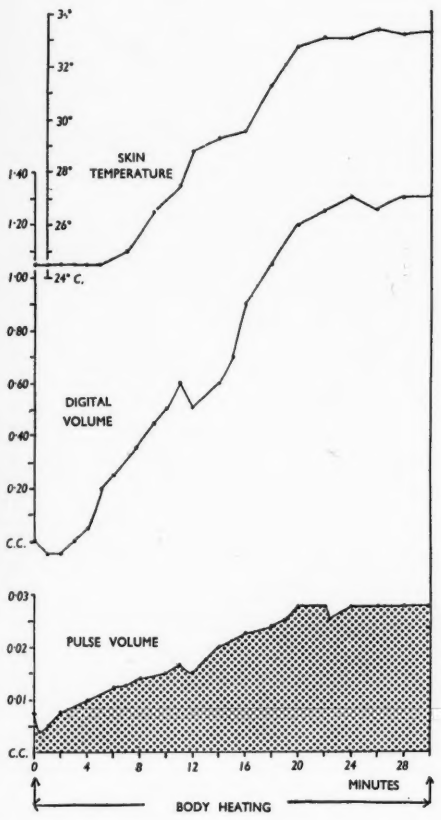

FIG. 55.—Chart prepared from a continuous record of the peripheral blood-flow during body heating in a normal subject, as used for differentiating between organic and functional diminution of the peripheral blood-flow.

Fig. 56 is representative of a typical case of arterial disease demonstrating the value of the method of assessing the degree of organic occlusion. It refers to a man 53 years of age with typical intermittent claudication in the calf of his left leg. In both limbs the pulse-volume at rest was extremely diminished and in fact not registrable. The blood-flow was therefore markedly diminished. In the right leg the pulse-volume increased with body heating, but reached only 0·01 c.c. (as against 0·02 c.c. normal) when fully dilated. We conclude that in this limb we are dealing with an organic arterial disease with marked superadded spasm. In the left limb the release of the central vasomotor tone did not result in an appreciable increase in the height of the pulse-volume. When fully dilated it reached only 0·003 c.c. (0·02 c.c. normal). We are therefore dealing with a limb in which the diminution in blood-flow is almost entirely organic in nature. Increased vasomotor tone does not appreciably contribute to the diminution in the blood-flow in this limb.

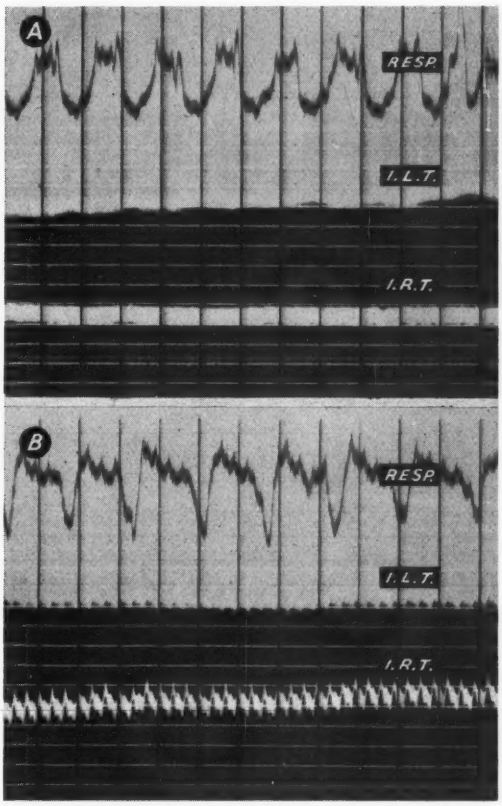

FIG. 56.—The effect of body heating on the pulse-volume in a typical case of arterial disease, affecting particularly the left leg. A, Pulse-volume (both big toes simultaneously recorded) during rest. Note pulse-volume completely absent in *both* limbs. B, Fully dilated. Pulse-volume in both limbs can now be recorded, but it remains below normal minimum vasodilatation level. Height of pulse-volume: 0·003 c.c. in the left and 0·01 c.c. in the right limb, as against 0·02 c.c. normal.

Plethysmography permits us to determine exactly whether the diminution in blood-flow in a case is organic or functional in type and allows us to assess the exact degree of both in a mixed case. It is important to note that in the case just reported there was definite organic interference in the blood-flow to that limb, which clinically did not yet give any signs or symptoms to that effect.

In man vasodilator nerves are probably not important. Vasodilatation is not an active mechanism, but merely the result of a decrease in central sympathetic vasoconstrictor tone. Reflex vasodilatation is therefore absent if the vessels have been disconnected from the centres by sympathectomy.

* Obviously the height of the pulse-volume depends also upon the volume of the digit enclosed in the plethysmograph. Therefore, strictly speaking, the pulse-volume should be corrected in each case to a mean digital volume (Goetz, 1946). For all practical purposes it is, however, sufficient to compare the uncorrected height of the pulse-volume of respective digits in various subjects if the above point is always kept in mind.

To give an exact example: in the patient from whom we obtained *Fig.* 57 a bilateral splanchnicectomy (Smithwick's operation) had been performed. On testing the circulation it was noted that the

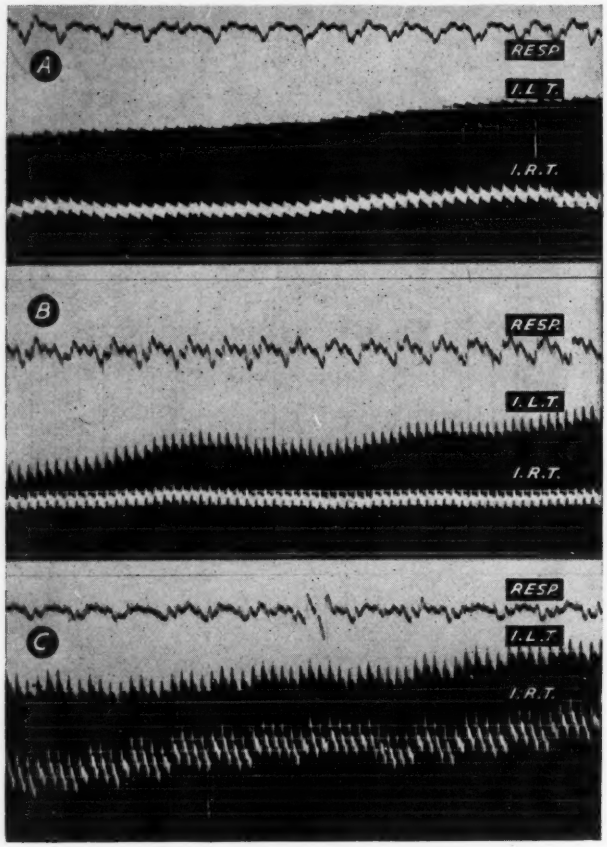

FIG. 57.—Plethysmographic tracing of the blood-flow simultaneously recorded in the two lower extremities, in a case which had undergone bilateral Smithwick's operation. A, Pulse-volume during rest. B, Pulse-volume after 30 minutes body heating. Note increase in the height of the pulse-volume of the left limb (upper record), but lack of response in the pulse-volume of the right limb. There was release of vasomotor tone in the left but not in the right limb. C, Following additional application of 10 minutes' local heat. Note increase in the pulse-volume of the right limb, indicating that the vessels are not organically occluded. For explanation *see* text.

vessels in the left limb dilated to body heating, i.e., the vessels were capable of reacting to central ablation of sympathetic tone and consequently could not have been sympathectomized. The vessels of the right limb remained completely inert and were therefore sympathectomized. It may be argued that the vessels in the right limb did not dilate because there was organic arterial disease. Such doubts can, however, easily be removed by applying local heat to the limb. The sympathectomized vessels still dilate on local application of heat, as indeed they did in this case (*Fig.* 57 C), the pulse-volume following that procedure reading 0·025 c.c. The failure of the vessels in the right leg to dilate was therefore functional and not organic in nature.

According to the clips fixed during the operation (*Fig.* 58), the second lumbar ganglion was removed on the right side, but was missed on the left side, the first being the lowest ganglion excised, confirming the plethysmographic appearances.

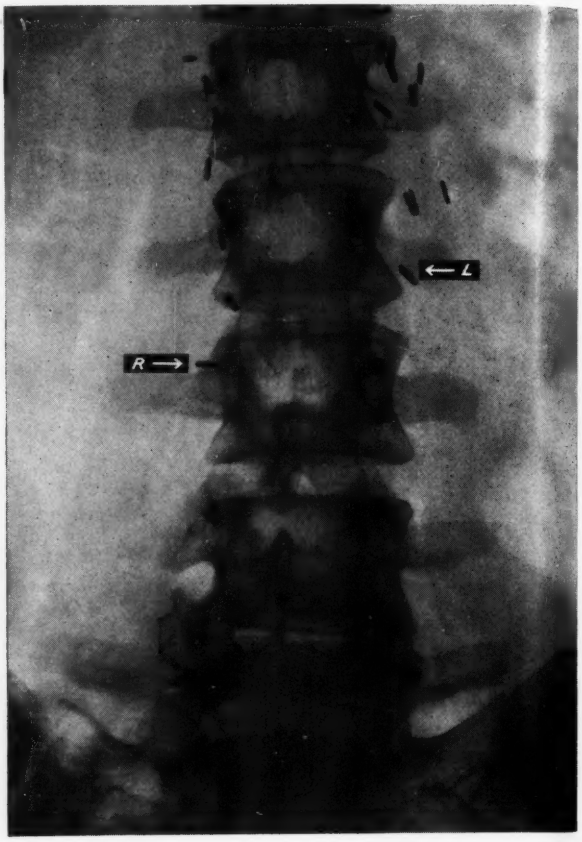

FIG. 58.—Radiograph of same case as *Fig.* 57. Note clips fixed at lower cut end of sympathetic. On the left the second lumbar ganglion has been missed, L1 being the lowest ganglion removed.

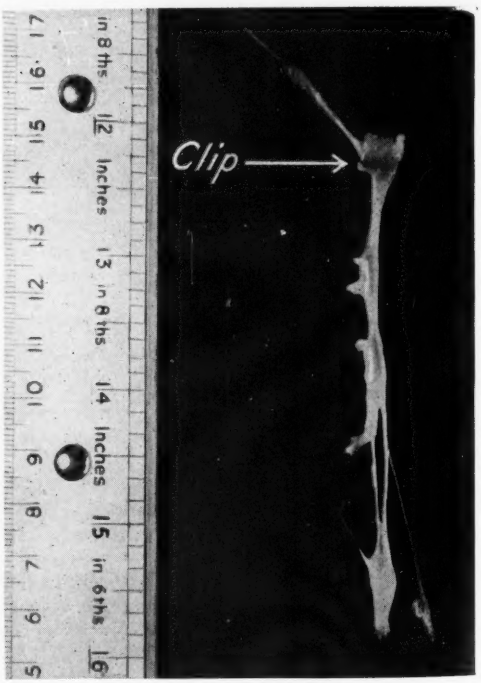

FIG. 59.—Specimen removed in same patient as in *Figs.* 57, 58. Note the silver clip on the top end of the specimen, and the large branch which went to the kidney.

Subsequent removal of the left second ganglion (*Fig.* 59) resulted in complete sympathetic denervation of this limb, as is well demonstrated in *Fig.* 60, when neither limb reacted to body heating. The value of plethysmography in determining the integrity

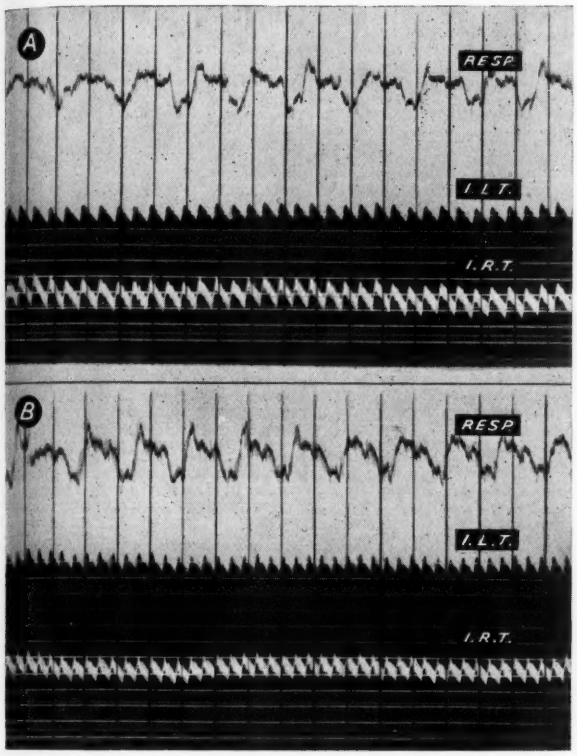

FIG. 60.—Plethysmograms of same case as in *Figs.* 57–59 but after additional sympathectomy on the left side. A, Pulse-volume in both lower limbs simultaneously recorded at rest. B, After 30 minutes' body heating. Note no increase in pulse-volume of either leg.

of the sympathetic pathways is extremely well illustrated by this example. Such information is most important in cases where, following sympathectomy, there has been a recurrence of symptoms, and where one is faced with the problem of determining whether this is due to a regeneration of the sympathetic pathways or to some other cause. It emphasizes the importance of determining in each case, immediately following operation, the completeness of the sympathetic denervation for future reference.

Dilatation from body heating is the result of inhibition of sympathetic activity produced by the return of heated blood from the immersed extremity acting on the thermosensitive centres in the hypothalamus. Dilatation is therefore not obtained if the blood-flow of the immersed limb has been interrupted either experimentally (*Fig.* 61) or by a pathological process (*Fig.* 62). There will be no, or an insufficient amount of, heated blood returned from such an extremity and body heating will fail to produce any or full vasodilatation of the vessels in the limb under examination. Unless we are aware of this fact we may wrongly conclude that the blood-vessels of the examined limb show organic disease while in fact their failure to dilate reflects the interference of the blood-flow of the immersed extremity and reveals nothing about the circulation in the examined limb.

A typical example of this nature is illustrated in *Fig.* 62. It refers to a young coloured male with completely normal cardiovascular system but who

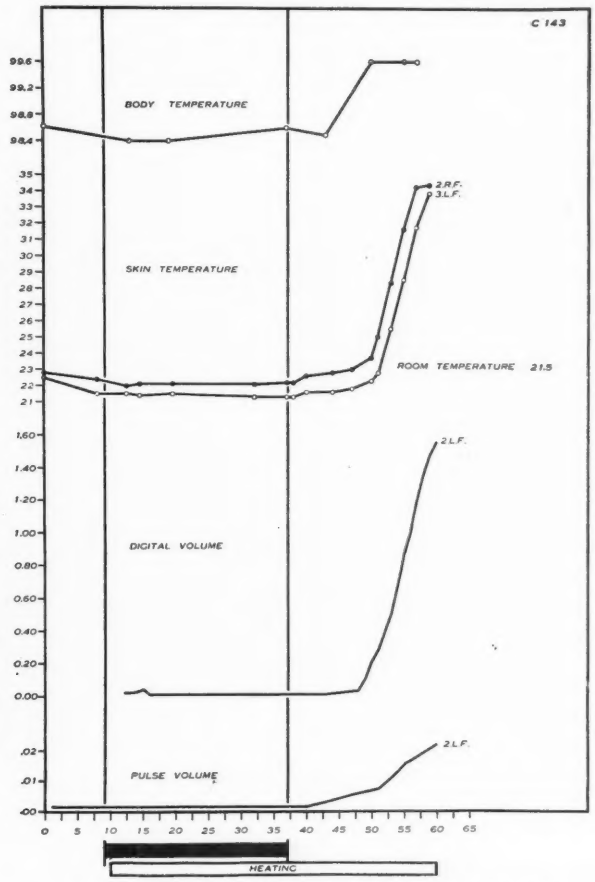

FIG. 61.—Failure of body heating to produce reflex vasodilatation while the blood-flow in the immersed extremity has been arrested experimentally. Black square indicates period of arrest of circulation to immersed limb by means of a blood-pressure cuff. Note prompt dilatation on release of the occluding pressure.

had his right axillary artery accidentally severed. The blood-flow in the other three limbs was completely normal. Body heating was carried out in this subject by immersing the left, and subsequently the right, arm in water of 45° C. for 30 minutes. The blood-flow in the right leg was tested. *Fig.* 62 (uninterrupted lines) gives the result of this test with reference to immersion of the left arm. After four minutes' body heating both the digital volume and the pulse-volume (first left toe) began to increase, and after six minutes the skin temperature (second left toe) followed. It took only twenty minutes for the pulse-volume in the right limb to reach values well above the minimum vasodilatation level (0·02 c.c.). The digital volume rose simultaneously for more than 1·00 c.c. and the skin temperature reached the normal vasodilatation level (34° C.) within 26 minutes. All values, therefore, indicated that the blood-flow in the right leg was completely normal. When the right (affected) arm was immersed the picture was vastly different (*Fig.* 62, interrupted lines). There was some dilatation of

the vessels in the right leg, but after thirty minutes' body heating the pulse-volume did not even register 0·01 c.c., the skin temperature reached 27° C. only, and the digital volume had risen only by 0·4 c.c. This, then, indicates clearly the importance of assuring that there is adequate return of heated blood in order to induce vasodilatation.

As already mentioned, the plethysmogram not only registers changes with each cardiac cycle

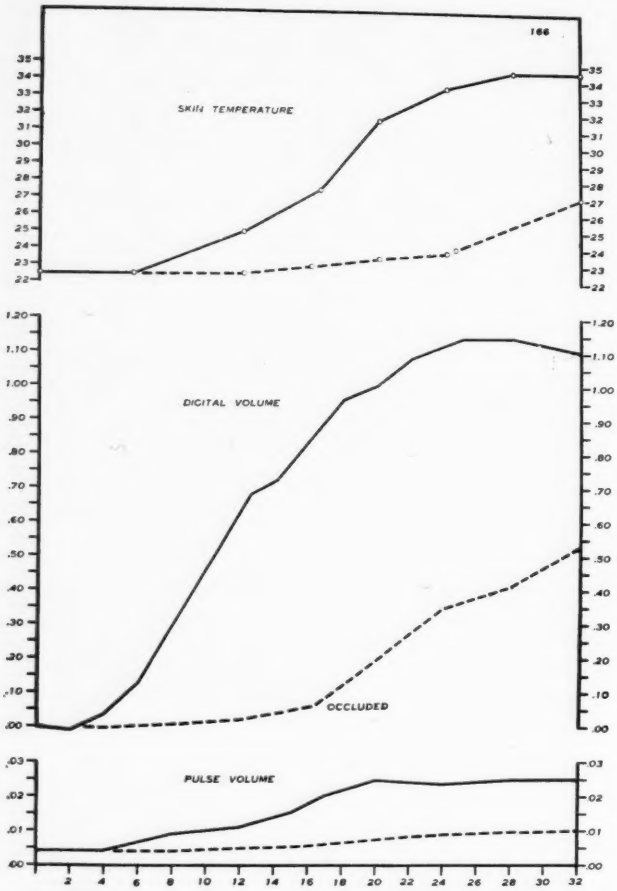

FIG. 62.—Graph prepared from continuous plethysmogram of big left toe. The digital volume, pulse-volume, and skin temperature were simultaneously recorded while body heating was carried out by: (*a*) immersing the normal left arm (uninterrupted lines); and (*b*) immersing the right arm with severed axillary artery (interrupted lines). Note failure to produce vasodilatation in the left leg from immersion of the right arm, although blood-flow normal as judged by the response to immersion of the left arm.

(the pulse-volume) but also fluctuations in the volume of the digit as a whole (*Fig.* 52 C). These changes in digital volume correspond to, and are mainly the result of, fluctuations in the height of the pulse-volume. Therefore any stimulus which decreases the height of the pulse-volume (*see* p. 28) also diminishes the digital volume. These changes in digital volume are no longer recorded in the sympathectomized limb and are therefore true reflex vasomotor phenomena. Conversely, their absence may be taken as proof that the sympathetic pathways have been interrupted, either accidentally or deliberately.

This is well illustrated by *Figs.* 63–65, which refer to a case which had undergone a cervicodorsal sympathectomy some thirty years previously (1917). The stellate and the second dorsal ganglion had been removed on the left side. To-day he presents with left-sided Horner's syndrome and anhydrosis

FIG. 63.—Minors' sweating test in a case which had undergone cervicodorsal sympathectomy (removal of the stellate and the second dorsal ganglion) in 1917 (!).

FIG. 64.—Same case as in *Fig.* 63, showing complete Horner's syndrome thirty-one years after cervicodorsal sympathectomy.

on the left side affecting the arm, the face, and the upper thorax (*Figs.* 63, 64). Plethysmographic examination reveals that the right hand reacts to a deep breath or the application of cold with marked vasoconstriction (*Fig.* 65). There is the normal decrease in pulse-volume and digital volume. In contrast the vessels of the left hand remain completely inert, neither the pulse-volume nor the

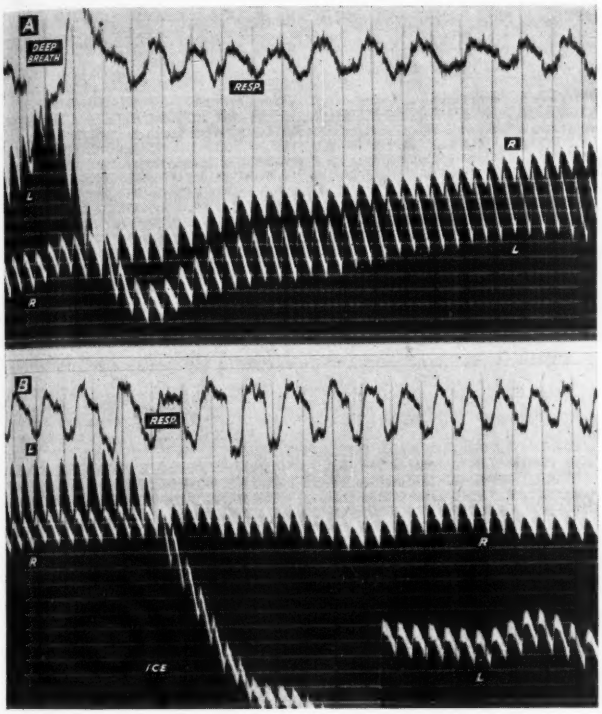

FIG. 65.—Plethysmogram of left and right third fingers simultaneously recorded in the case illustrated in *Figs.* 63, 64, i.e., thirty-one years after cervicodorsal sympathectomy. Note absence of vasoconstriction in the sympathectomized left finger to a deep breath in A and to immersion of both feet into ice-cold water in B.

digital volume showing any signs of constriction. It is therefore evident that the vessels in the left hand are disconnected from the higher centres. Sympathectomy is still complete in respect to the vasomotor, sudomotor, and oculopupillary sympathetic phenomena after thirty-one years!

Various authors have investigated the completeness of sympathetic denervation by means of sweating tests, and from the presence or absence of sweating have drawn conclusions as to the integrity of vasomotor pathways. Although often both do go together the conclusion is not always permissible, and we have cases on our record where sweating returned in a sympathectomized area but vasomotor pathways could not be demonstrated (Goetz, 1948, b). In order to assess the various functions of the sympathetic each has to be tested separately, otherwise we lay ourselves open to grave mistakes.

At this stage we may pause for a moment to ask ourselves the question: In which respect is plethysmography superior to skin temperature readings, the method most commonly used in the clinic to-day? I have already pointed out that skin temperature readings are capable of furnishing us with an indirect index of the peripheral blood-flow only. Plethysmography is a quantitative method. The difficulties met with in standardizing the conditions under which the skin temperatures have to be taken to give comparable values have been pointed out. Heat production and, particularly, heat dissipation are governed by many variables

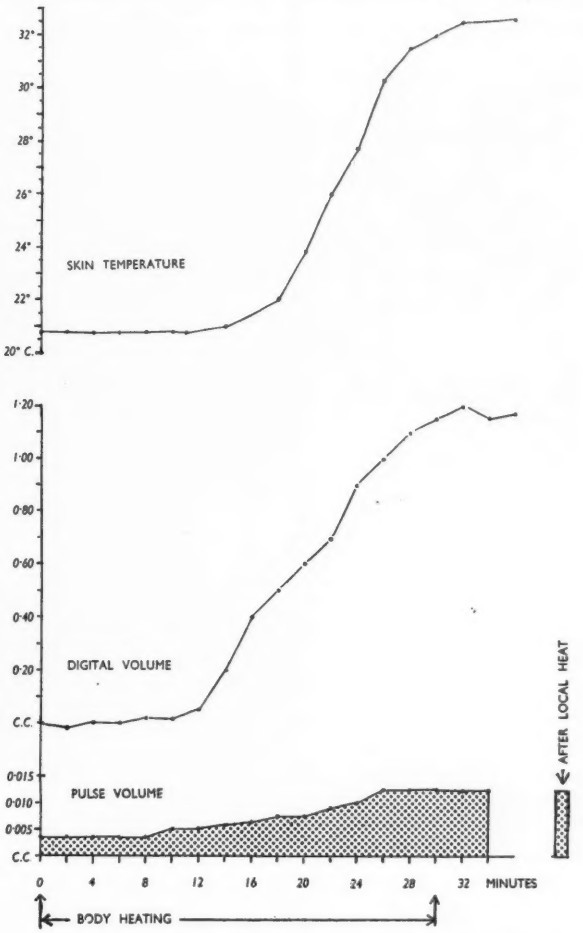

FIG. 66.—Effect of body heating upon the pulse-volume, digital volume, rate of blood-flow, and the skin temperature in a case which presented with Raynaud's phenomenon. Note the diminished response of the pulse-volume, indicating organic interference to the digital blood-flow, but normal response of the skin temperature indicating normal blood-flow. For explanation *see* text.

difficult to control, especially if the tests are carried out in different wards, the theatre, or in the patient's home. A rise in skin temperature of e.g. 5° C. may be produced by a considerable or negligible increase in blood-flow. The different (often most unphysiological) techniques adopted in taking skin temperatures by the various workers in this field may yield very different results even in the same patient. In the past, skin temperatures have been interpreted in terms of blood-flow by assuming that the temperature of the arterial blood in each case is of the same, i.e., body, temperature. The work of Bazett and his school on the precooling of the arterial blood, however, does throw all older ideas about the interpretation of skin temperature readings out of gear. The fictitious assumption underlying the evaluation of the skin temperature that the blood in the arteries is of body temperature can no longer be maintained.

Bazett has clearly demonstrated that the blood temperature of the radial artery may at times be as low as 20–25° C.! This precooling of the arterial blood is of particular importance in cases in which the blood-flow is carried on by the collateral circulation and these cases form a great percentage of our patients; it must affect skin temperatures in these cases considerably. We do know that the collateral

FIG. 67.—Microphotograph of one of the digital arteries of the digit examined in *Fig.* 66. Note that the histology supports the plethysmographic appearances.

vessels as a rule take a very superficial course (*Figs.* 72, 74), making their blood particularly liable to precooling. It stands to reason that under such a condition the skin temperatures are almost, if not absolutely, valueless unless due allowance for the precooling of the blood is made. The difficulties of translating skin temperatures into rate of blood-flow are therefore considerable and skin temperature readings may be actually misleading.

All these objections do not apply to plethysmography. But apart from the above considerations we could demonstrate that even a ' normal ' response of the skin temperature to body heating does not necessarily mean that we are dealing with normal blood-vessels, and the following set of pictures are illustrated to demonstrate how plethysmography is capable of picking out minor changes in the peripheral vessels at a time when the skin temperature readings still give normal values, i.e., a rise to 32° C. or above on body heating (*Figs.* 66, 67).

The following was a case of Raynaud's phenomenon. On testing, the pulse-volume reached only 0·012 c.c. during full dilatation, as against 0·02 c.c. normal. The skin temperature, however, still reached 32° C. with body heating on various occasions (*Fig.* 66). The patient succumbed to an intercurrent disease which had nothing to do with the vascular pathology, and we had the opportunity of sectioning the vessels of the digits previously examined. While the skin temperatures had suggested the vessels to be normal, judged by the plethysmographic readings there should have been organic arterial disease. *Fig.* 67 shows the section of one of the two digital arteries, demonstrating considerable organic interference to the lumen, proving the correctness of the plethysmographic appearances. Incidentally, the radial artery was completely normal, as is the rule in patients afflicted with Raynaud's phenomenon, illustrating our contention that oscillometry is unable to furnish us with a concept of the state of the *peripheral circulation.*

THE ESTIMATION OF THE RATE OF ARTERIAL INFLOW AND OF THE COLLATERAL CIRCULATION BY MEANS OF PLETHYSMOGRAPHY

In addition to what has been said above, the plethysmogram permits measurement of the actual rate of blood-flow through the digits, by means of the so-called venous congestion test. In brief, the test consists in arresting the venous return from the digits by means of a blood-pressure cuff fixed at the ankle, the wrist, or on the digit itself, without impeding the arterial inflow.* On applying the pressure, the digital volume increases with each heart beat, first along a straight line (*Fig.* 68), but as the vascular bed of the periphery fills, the tracing slopes gradually towards the horizontal and eventually runs parallel to the base-line. When the pressure is released the tracing returns, as a rule, to the original level. During the first part of the tracing the rise in digital volume is proportional to, and permits the calculation of, the arterial inflow. The results obtained with this method are very consistent and enable evaluation with great accuracy as long as the pressure of the cuff is well below the diastolic pressure and above 30 mm. Hg.

In the normal the rate of arterial inflow depends mainly upon the height of the pulse-volume, i.e., upon the vasomotor tone. The higher the pulse-volume, the greater the arterial inflow. A typical example of the venous congestion test in a normal individual under varying states of vasomotor tone is given in *Fig.* 68. With vessels fully constricted and a pulse-volume of 0·002 c.c. only, it takes the digital volume ten seconds to increase by 0·01 c.c. As the vessels relax, and the pulse-volume increases (to 0·010 c.c.), the arterial inflow reaches two-hundredths of a c.c. in two seconds (*Fig.* 68 B). With further dilatation and a pulse-volume of 0·015 c.c., the arterial inflow amounts to 0·06 c.c. for two seconds. There occurred therefore a thirty-fold increase in the

* The site of the congesting cuff is of importance for the calculation of the arterial inflow. In order to get an absolute and direct measure of the arterial inflow, the cuff should be fixed at the base of the digit itself. This is possible when dealing with the fingers, but impracticable with the toes. It has been found that the gradient of the tracing is steeper and the rate of blood-flow calculated three times greater if the cuff has been fixed at the base of the fingers than if it had been fixed at the wrist. This factor has therefore to be taken into account when calculating the rate of blood-flow from tracings obtained with the cuff fixed at the wrist or the ankle. The whole question has been fully discussed in earlier papers (Goetz, 1943, 1946).

rate of blood-flow. The rate of blood-flow in normal subjects thus calculated varied between 1 c.c. during full constriction and 90 c.c. per minute for 100 c.c. tissue when fully dilated (Goetz, 1943, 1946).

In assessing this wide range in the adjustment of the peripheral blood-flow we have to remember that, according to Poiseuille's law, the rate of flow varies as the fourth power of the radius of the vessel. In quoting this law I am fully aware of the fact that it was first elaborated for rigid tubes. However, subsequent workers have demonstrated that it is

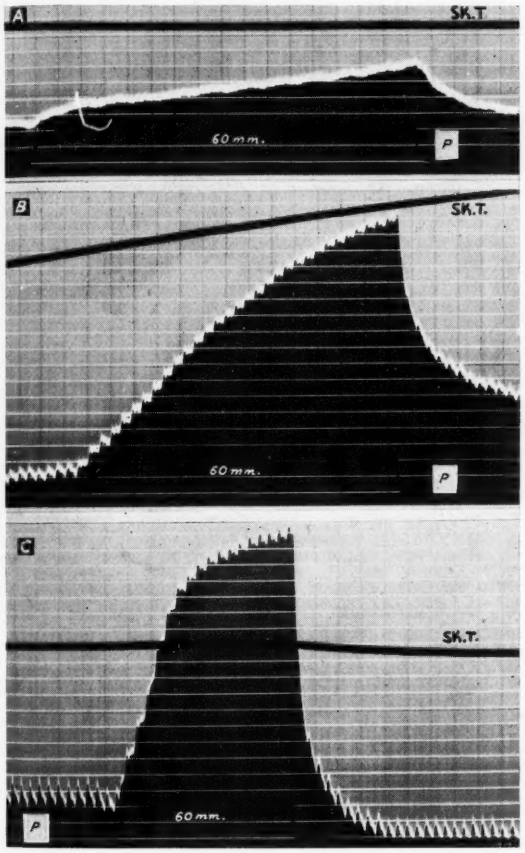

FIG. 68.—Venous congestion tests during various states of vasomotor tone. A, Constricted; B, During dilatation; C, Fully dilated. P, Record of pressure in cuff; Sk.T., Skin temperature.

applicable in man, with minor modifications only. Therefore, if the vessel increases its internal diameter from 1 mm. to 2 mm. (that is, to twice its size), the blood-flow will be increased 16 times! Or, in order to get an increase in blood-flow of 80 times, the diameter of the vessel has to increase only from 1 mm. to 3 mm. Vice versa, knowledge about this law furnishes us with a conception how a minor interference to the lumen of the vessel will produce such large effect on the blood-flow. It explains, too, the sudden diminution in the circulation occurring from one day to the next, so often elicited in the patient's history.

Though in general there is a direct relation between the height of the pulse-volume and the gradient of the arterial inflow on venous congestion, careful scrutiny of a large number of tracings does demonstrate that this is not always the case. This is well illustrated by *Fig.* 69, obtained in a normal individual during body heating. Between *Fig.* 69 A and B, twenty minutes' body heating elapsed. On comparing the two, a marked increase both in digital volume and the rate of blood-flow is recorded.

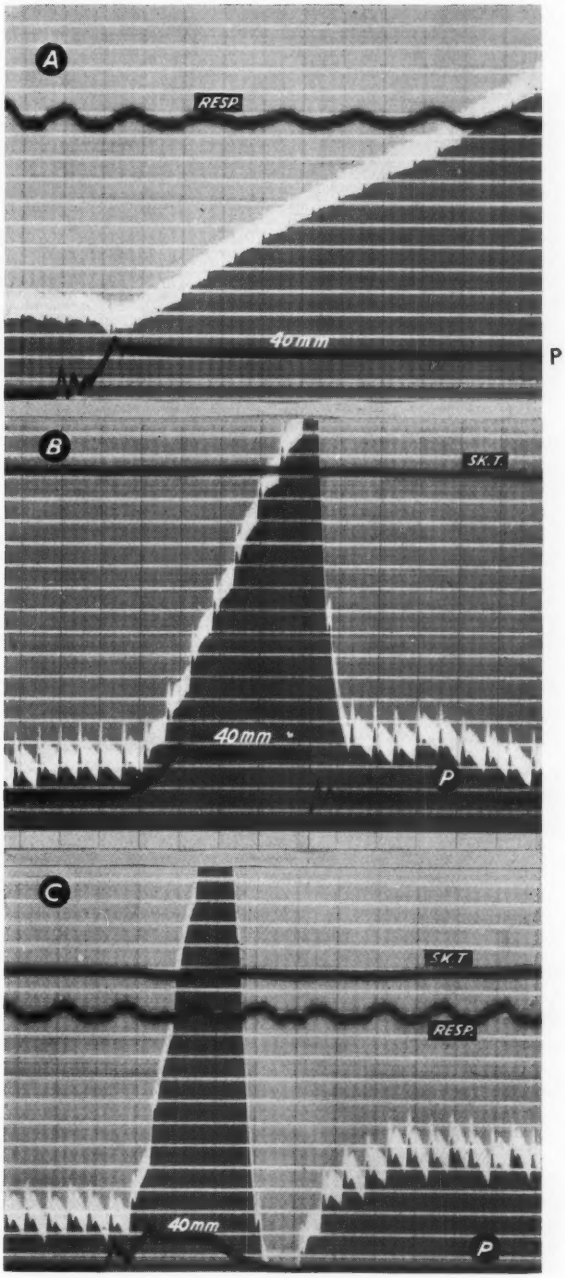

FIG. 69.—Venous congestion test in a normal individual during various states of vasomotor tone. A, During rest; B, During dilatation; C, Fully dilated. Note opening of the collaterals between B and C. For explanation *see* text.

There is release of vasoconstrictor tone. *Fig.* 69 C was obtained ten minutes later. Although no further increase in digital volume has been obtained, a marked additional increase in the rate of blood-flow is noted. This further increase is due to the opening of small collateral vessels in which the blood-flow is

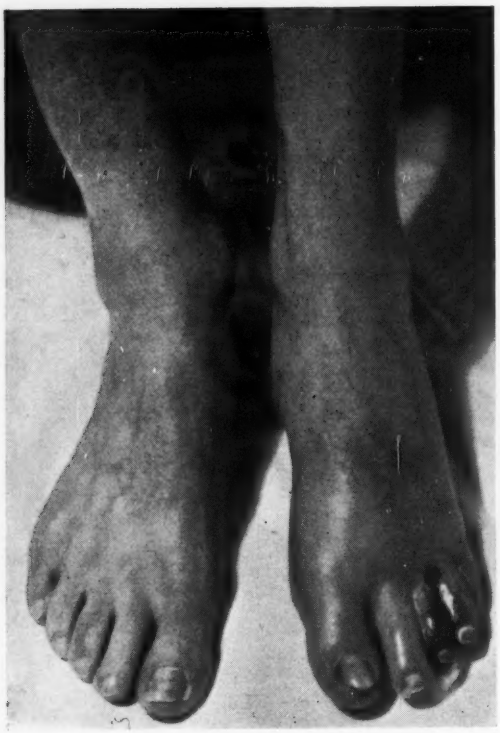

FIG. 70.—Photograph of a typical case of thrombo-angiitis obliterans.

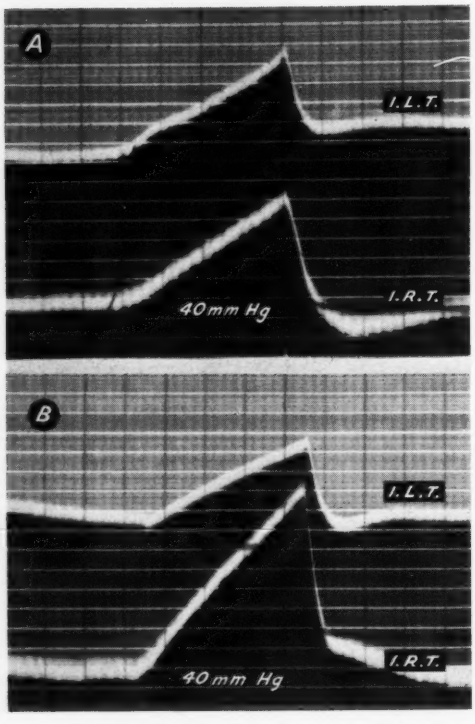

FIG. 71.—Venous congestion test in a typical case of thrombo-angiitis obliterans. A, At rest; and B, Fully dilated. Both limbs simultaneously recorded. Note absence of pulse-volume in either leg both during rest and after body heating. Although there is no increase in the pulse-volume of the right leg between A and B, a definite increase in the rate of the arterial inflow is recorded. There is complete organic occlusion of the main vessels in both limbs, with a poor collateral circulation in the left, and a reasonably good collateral circulation in the right, limb.

continuous without pulsating, and hence does not contribute towards the height of the pulse-volume (Goetz, 1946).

This finding is not only of academic interest but of considerable clinical importance, since it permits exact measurements of the collateral circulation in cases of peripheral vascular disease, as is well illustrated by the following cases.

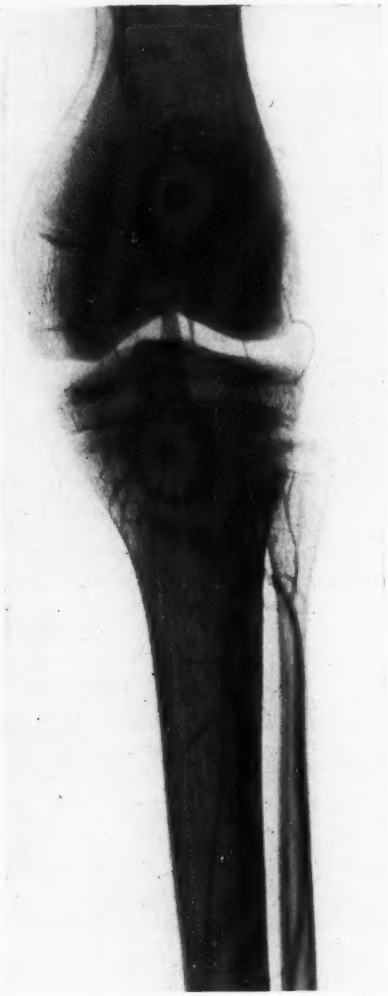

FIG. 72.—Arteriogram showing embolus in the popliteal artery. Note filling of both tibial arteries via the collaterals.

Figs. 70, 71 were obtained in a typical case of thrombo-angiitis obliterans and in which there was complete organic occlusion of the blood-flow in both limbs as judged by the fact that the pulse-volume was not registrable either during rest or during full vasodilatation. Measurement of the arterial inflow by means of the venous congestion test gave, in the left leg, identical and markedly diminished values both during rest and after body heating. In the right leg, however, there is a definite increase in the rate of arterial inflow on body heating, which increase is due to the opening of the collateral circulation. By means of plethysmography we are therefore capable of measuring the degree of the collateral circulation exactly. Such information is of paramount importance in assessing the prognosis, planning the therapy

in general, and advising for or against sympathectomy in a particular case on a scientific basis.

The value of the method in measuring the collateral circulation is further illustrated by the following two cases: *Figs.* 72, 73 refer to a native male, 16 years of age, with an established diagnosis of mitral stenosis and aortic incompetence complicated by subacute bacterial endocarditis. He was

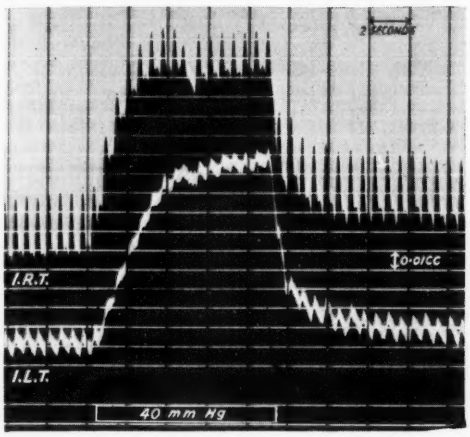

FIG. 73.—Plethysmogram and venous congestion test (fully dilated) in both limbs simultaneously recorded in the same case of popliteal embolism as in *Fig.* 72. Note the excellent rate of arterial inflow in the left limb due to the good collateral circulation. For explanation, *see* text.

referred to us for sudden pain in the left calf, where the leg became swollen and tender. On examination the popliteal artery was pulsating in its most proximal course, but a sudden stop could be elicited about the middle of the popliteal space where the artery felt thickened and surrounded by inflammatory tissue. The peripheral vessels could not be felt at this stage, although months later the dorsalis pedis became palpable. None of the collateral arteries around the knee-joint was palpable. There was slight cyanosis of the limb, which, however, did not feel unduly cold to touch. No signs of any venous involvement were detected. There was no doubt that an embolus had lodged in the popliteal artery.

The arteriogram (*Fig.* 72) shows an abrupt block at the bifurcation of the popliteal artery. The collaterals described around the knee in the anatomical texts are outlined. A point made previously (Goetz, 1945) is of some considerable importance: in Nature's attempt to develop the collateral circulation, the blood tries to re-enter the main vessels at a point beyond the occlusion. This is well demonstrated in this plate, and the main vessels beyond the occlusion, i.e., the anterior and posterior tibial arteries, are perfectly filled with the contrast medium. This is one of the reasons for instituting anticoagulant therapy and why the prognosis is good in a young person if the embolism occurs at this site. The blood can re-enter via the collaterals into healthy main arteries. By using a long exposure during arteriography (Goetz, 1945), it was possible to outline the arteries above and below the occlusion, as well as the collaterals, without difficulty on one film.

Plethysmographic tracings recorded a pulse-volume in both lower limbs. However, in the left limb it reached only one-quarter of that of the normal limb, i.e., 0·008 c.c. as against 0·035 c.c. On recording the arterial inflow by means of the venous congestion test, the actual blood-flow through the periphery is not diminished to the degree expected

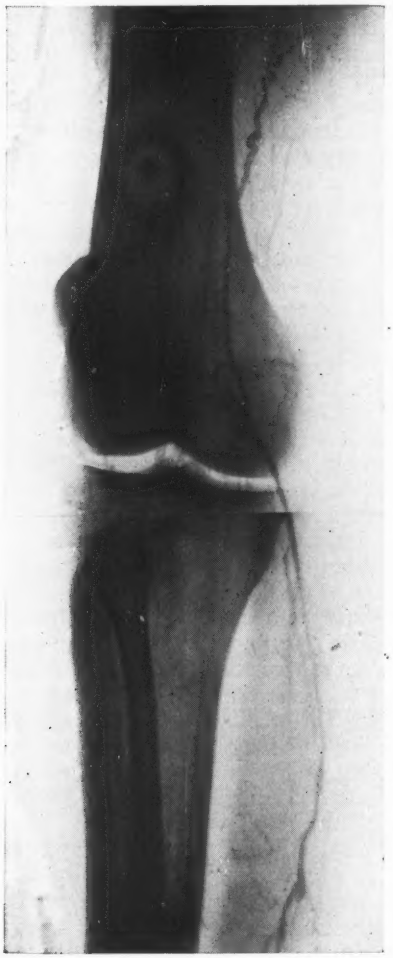

FIG. 74.—Arteriogram showing complete occlusion of the femoral artery and good collaterals.

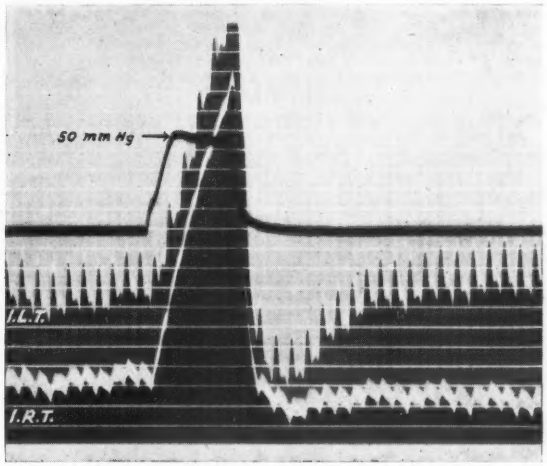

FIG. 75.—Venous congestion test in the same case as *Fig.* 74, both limbs simultaneously recorded. Note the discrepancy between the height of the pulse-volume in the two limbs and the arterial inflow. The arterial inflow is infinitely better than would be suggested by the height of the pulse-volume and is due to the good collateral circulation. For explanation, *see* text.

from the difference in the height of the pulse-volume (*Fig.* 73). Indeed the arterial inflow in the two limbs, simultaneously recorded during a venous congestion test, compares as 5 : 3. The blood-flow in the affected leg, therefore, is diminished by 40 per cent only. From the arteriogram we know that the main artery is completely blocked and that the circulation is being carried on by the collaterals only. Some of them are wide enough to permit a pulse to be transmitted into the periphery ; however, in the majority of them the blood-flow is continuous and therefore is not reflected in the pulse-volume, but contributes towards the rate of arterial inflow, which consequently is larger than to be expected from the height of the pulse-volume.

Figs. 74, 75 refer to a European male, 27 years of age, who presented with typical signs of early intermittent claudication in the left leg, which had always been cold and worrying. This had come on during the past three years and had remained relatively stationary during that time. He could walk about 400–500 yd. on the level, after which he would develop discomfort. He had never been ill. There was no history of migrating phlebitis or any disease to which the arterial disease could be secondary. On general examination nothing was found except absent popliteal, dorsalis pedis, and posterior tibial arteries on the affected side. The heart, blood-pressure, and central nervous system were all normal. The arteriogram (*Fig.* 74) revealed that the femoral artery was completely occluded and the circulation was carried on by collateral vessels only, which were extremely tortuous and elongated.

The plethysmogram in this case (*Fig.* 75) is most striking and illuminates the value of plethysmography as a method of assessing the peripheral blood-flow extremely well. Although the femoral artery is completely occluded, a pulse-volume of 0·008 c.c. is recorded during full dilatation, as compared with 0·028 c.c. in the normal limb. The venous congestion test reveals that despite this difference in the height of the pulse-volume, the arterial inflow in both limbs is for all practical purposes equal. This is due to the development of a large network of collaterals. On the strength of these findings we suggested sympathectomy and could offer confidently a good prognosis.

SUMMARY

In summarizing I would like to stress the following points :—

The results that are obtained with our portable digital plethysmograph are not only referable to the blood-flow through the skin but are also representative of the peripheral circulation in a strict sense.

The method registers the blood-flow accurately under most physiological conditions and furnishes a record which is standardized. The results, therefore, can be compared from patient to patient.

The method lends itself easily to routine clinical investigation, in the ward, the operating theatre, or at the patient's home.

The changes in pulse-volume not only serve as an important guide in distinguishing between a functional diminution in blood-flow (a high vasomotor tone) and an organic arterial disease, but permit the assessing of the part played by each in a mixed case.

In conjunction with the venous congestion test the method furnishes us with exact information of the arterial inflow and an accurate concept of the collateral circulation, thus providing us with a scientific approach to the diagnosis, treatment, and prognosis in diseases of the peripheral vascular system. The value of this information is illustrated in a number of cases.

It is in the early and doubtful case when plethysmography is most helpful in confirming or excluding the existence of an arterial lesion. At that stage skin temperature readings may still give normal values, with the result that many an early case is missed unless we resort to plethysmography.

Mention is made of the difficulties involved in translating skin temperatures into rate of peripheral blood-flow.

Successful treatment depends to a large degree upon the correct diagnosis in the incipient stages of the disease. Plethysmography therefore is not only an important adjunct in the investigation of peripheral vascular diseases, but also points the way to a more rational approach in their treatment.

The importance of measuring the peripheral (ultimate) blood-flow in contrast to the penultimate circulation has been stressed. It is pointed out that the danger to the patient's limb or life does not come from a deficiency of the blood-flow through the muscles, but from interference to the blood-flow through the skin, i.e., not from intermittent claudication but from threatening or developed gangrene. It is for these reasons that exact evaluation of the ultimate blood-flow in each case of arterial disease is of such importance, irrespective of the fact whether he does or does not present with signs and symptoms in reference to the blood-flow through the skin.

A CASE OF GASTRIC CARCINOMA WITH SPREAD EXCLUSIVELY TO THE REMAINDER OF THE BOWEL AND PERIANAL SKIN

By GEORGE LUMB, London

It is considered of importance to record the following case of gastric carcinoma in view of its very unusual clinical history, and the remarkable pathological picture of spread exclusively confined to the entire length of the bowel including the anus and perianal skin. Of this last feature no other example has been traced in a search of the literature.

Spread from carcinoma of the stomach to the remainder of the bowel with the production of areas of thickening and stenosis is known to occur infrequently, but relatively few cases are on record. Thus Dixon and Stevens (1936) were able to collect only 37 cases from the literature whilst reporting 6 new examples.

Since that time Judd, Larsen, and Tilden (1939) have reported 1 case, Saphir and Parker (1943) have described a further 5 cases, and Collins (1946) has reported 1 case.

CASE REPORT

History.—E. J., female, aged 59 years, under the care of Mr. E. Stanley Lee. First admission Sept. 20, 1946, complaining of six weeks' duration of constipation

Fig. 76.—Barium meal showing constant filling defect in ascending colon.

and ribbon-like stools, together with a constant desire to defæcate. Sigmoidoscopy at that time showed the mucous membrane of the anal canal and rectum to be granular and œdematous. The rectal wall was rigid. Urine normal. W.R. and Kahn reaction negative. Gonococcal complement-deviation test negative. Frei test negative. Stools negative for ova and amœbæ. Barium enema showed narrowing of a short length of ascending colon with a constant filling defect. A radiological and clinical diagnosis was made of carcinoma of the ascending colon, and the rectal condition was thought to be inflammatory and independent (*Fig.* 76). Operation by Mr. Lee, Nov. 19, 1946. No tumour was felt in

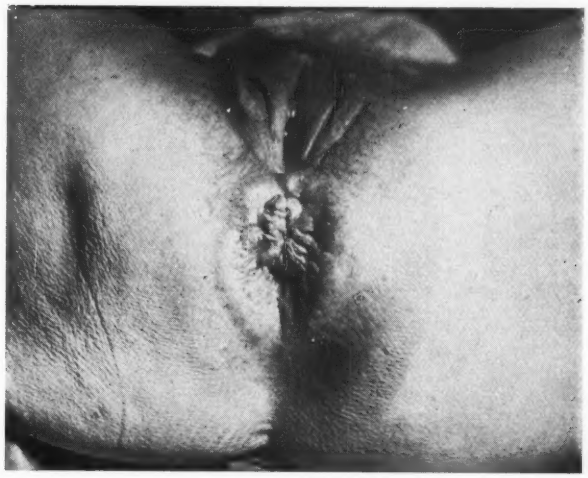

Fig. 77.—Thickening and infiltration of perianal skin.

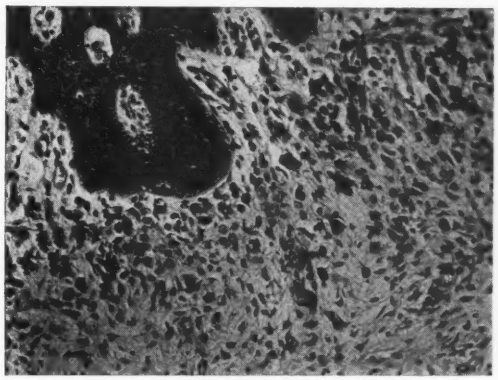

Fig. 78.—Biopsy before death of perianal skin showing cellular infiltration. (× 36.)

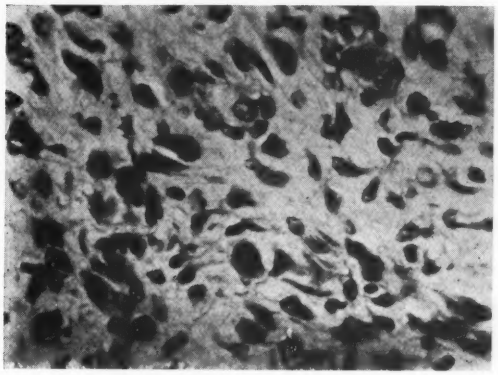

Fig. 79.—High-power view of same biopsy as *Fig.* 78. (× 108.)

the colon, but at operation some rubbery thickening in the ascending and transverse colon could be palpated. A right hemicolectomy was decided upon with side-to-side

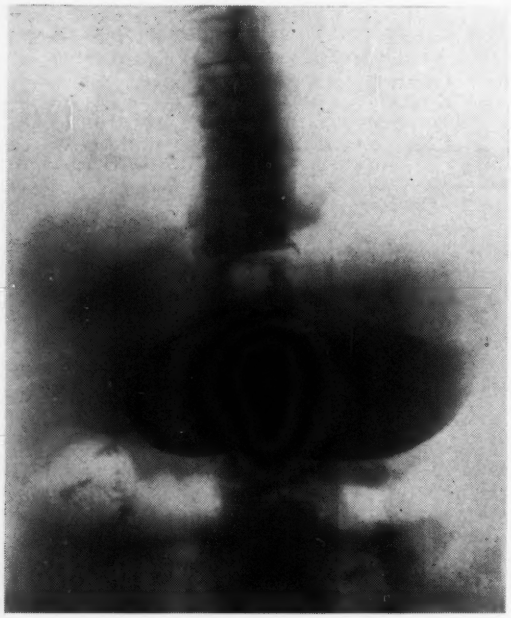

FIG. 80.—Barium meal to show pyloric stenosis (24-hour film).

anastomosis. Morbid anatomy and microscopy of the operation specimen showed no evidence of neoplasm or other abnormalities. The areas of thickening apparent at operation were not demonstrable in the fixed specimen, and in a re-examination of these specimens in the light of the subsequent diagnosis, no malignant cells could be discovered. Discharged with wound healed Dec. 7, 1946.

Second admission May 1, 1947, complaining of mucous diarrhœa with some incontinence of fæces; vague pain in the right abdomen and general malaise. On examination a slightly tender lump was palpated in the right iliac fossa; induration, feeling like a fibrous plaque around the anus spreading up the anal canal into the rectum, was observed (*Fig.* 77). By June 20 considerable ascites had developed. Investigations—Ascitic fluid showed no malignant cells. Frei test repeatedly negative. Leucocyte count 7700 per c.mm., polymorphs 76 per cent, lymphocytes 19 per cent, eosinophils 3 per cent. Radiograph of chest—normal. Biopsy of rectum and anal region was performed on July 3. Microscopy showed no abnormality in the rectal fragments. The skin from outside the anus and the anal canal itself showed the corium to be infiltrated with large deeply staining cells, many of which were binucleate. Considerable fibrous tissue increase was noted. The general appearances were those of a chronic granulomatous condition. The possibility of a reticulosis of malignant type was considered (*Figs.* 78, 79). Progress: July 10, 3 pints of ascitic fluid were withdrawn. July 20, laparotomy by Mr. Lee with the intention of performing a terminal ileostomy. Four pints of clear fluid were found in the abdominal cavity. The surface of the small bowel was purple in colour, with many dilated vessels on its surface. Intermittent, hard, contracted white areas causing partial gut obstruction were seen in the ileum and at the pylorus. The patient's condition began to deteriorate steadily, with considerable loss of weight, vomiting, recurrent ascites, and diarrhœa. After tapping the ascites ill-defined abdominal masses could be palpated. A barium meal on Oct. 9 showed almost complete pyloric obstruction (*Fig.* 80). Mr. Lee again opened the abdomen on Oct. 13, when he found a hard plaque at the pylorus. He performed an anterior gastro-enterostomy. Vomiting thereafter ceased, but diarrhœa and incontinence increased.

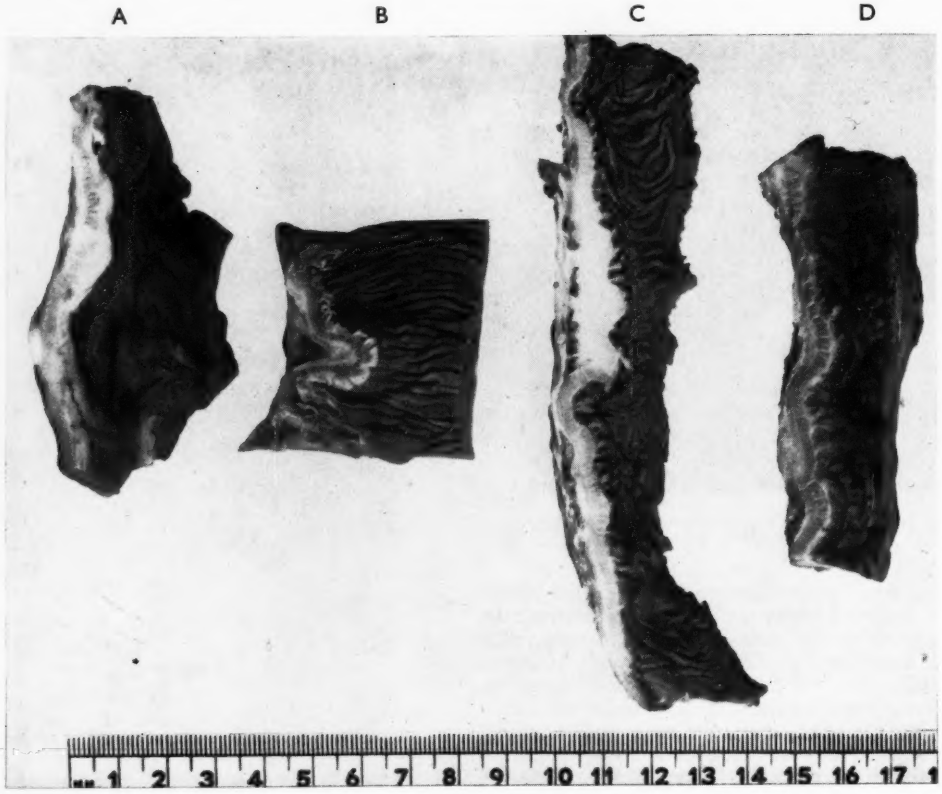

FIG. 81.—Post-mortem specimens of: (A) Stomach, showing polypoid area; (B) Jejunum; (C) Colon; (D) Rectum.

The patient went slowly downhill, becoming grossly wasted, and died on Dec. 2.

POST-MORTEM EXAMINATION (Dec. 3).—The body of a grossly emaciated female. Skull and contents, mouth, neck, larynx and pharynx, heart, lungs, liver, gall-bladder, spleen, kidneys, pancreas, genital organs, and bladder were all normal. No free fluid in the abdominal cavity. No evidence of peritoneal metastases. The omentum

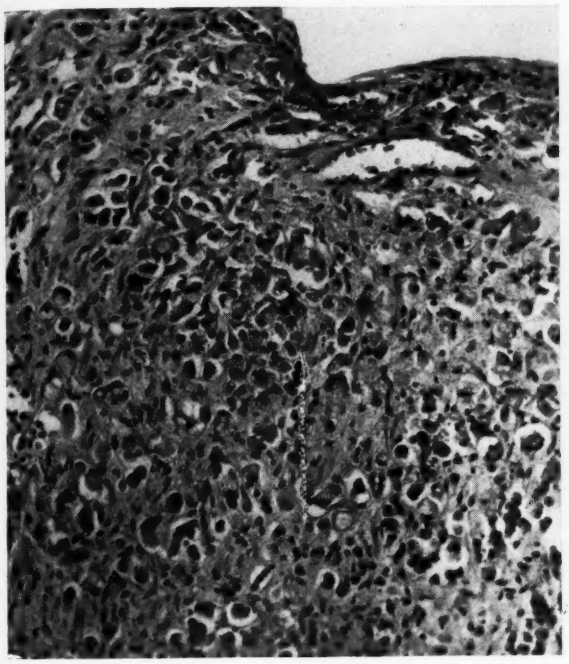

FIG. 82.—Stomach tumour, showing anaplastic nature of growth. (× 60.)

was rolled up but not invaded by carcinoma. No enlarged mesenteric or other abdominal lymph-nodes. The thoracic duct was normal. The stomata of the hemicolectomy and gastro-enterostomy were patent. The lower end of the œsophagus was thickened. Generalized thickening of the wall of the stomach was noted, which was more marked at the pyloric end, where the mucosa was somewhat polypoid, having an appearance similar to that of cerebral convolutions. Jejunum and ileum showed generalized bowel thickening, together with fibrous constrictions at intervals, causing gross diminution of the lumen. The colour of the entire bowel was bluish-black.

There was generalized thickening of the wall of the colon and rectum with fibrous constrictions at intervals similar to those seen in the small bowel. The mucous surface of the entire bowel was pale and smooth, with the exception of the pyloric end of the stomach and the rectum, which were somewhat polypoid and reddened in colour (*Fig.* 81). No ulceration was seen at any point. In the anal canal and the skin around the anal orifice for a diameter of 6 cm. there was dense fibrous thickening and induration (*see Fig.* 77).

MICROSCOPY.—Only the gastro-intestinal tract will be described in detail. There was no evidence of metastases in any other situation, including the liver, lungs, pleura, and ovaries. The abdominal lymph-nodes and the thoracic duct were normal.

The stomach showed diffuse thickening of all coats. The peptic cells of the mucosa were atrophied, and transformation to cells of intestinal lining type, with numerous goblet cells, was a noticeable feature.

In the pyloric region, the mucosa was replaced by sheets of carcinoma cells, the majority of which were round in shape and many contained mucus in their cytoplasm, some being of ' signet ring ' type. Mucinous material was present in the matrix between the cells. There was considerable anaplastic cellular variation, from those containing mucus to small round cells with deeply staining nuclei and clear pink cytoplasm. The cells were mostly separated from one another in a very loose matrix (*Fig.* 82).

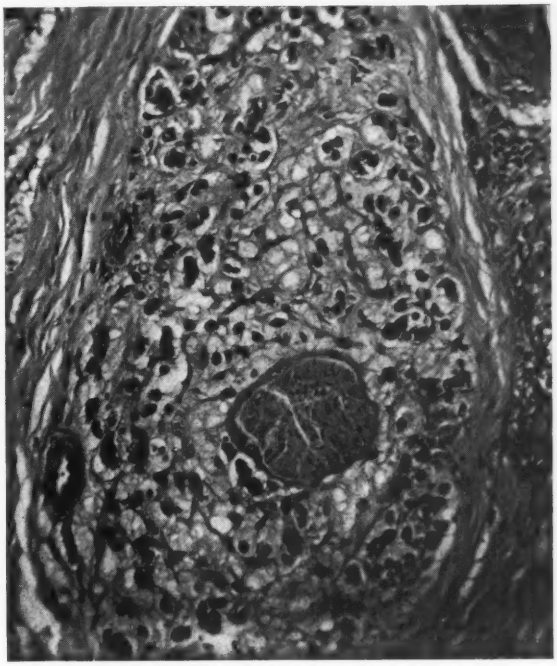

FIG. 83.—Infiltration around nerve by mucoid cells. (× 60.)

Streams of malignant cells were seen penetrating the submucosa and muscularis, passing into the subserosa. Infiltration around nerves was common (*Fig.* 83), and the majority of the small submucous lymphatics were seen to be plugged with malignant cells (*Fig.* 84).

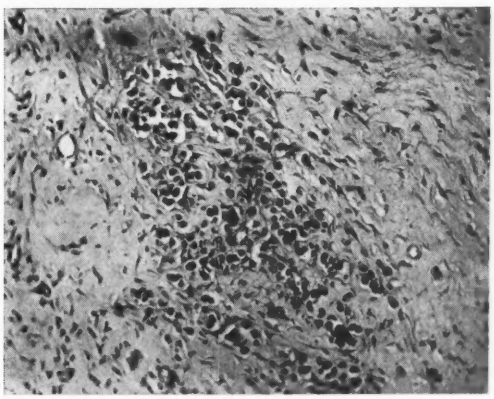

FIG. 84.—Blocking of submucous lymphatics by tumour cells. (× 36.)

Considerable overgrowth of fibrous tissue was seen in the submucosa and subserosa, together with extensive infiltration of the muscularis.

Malignant cells in smaller numbers were seen throughout the wall of the stomach and spread into the surface layers of the lower œsophagus had occurred.

Throughout the remainder of the bowel as far as the rectum carcinomatous infiltration and fibrosis could be

traced, and in all situations the submucous region was markedly affected. In the rectum carcinomatous infiltration of the muscularis was particularly well marked (*Fig.* 85).

In the colon blocking of the serous and subserous lymph-channels with carcinoma cells had occurred in addition to the submucous lymphatic plugging described above. No evidence of metastasis via the blood-stream was demonstrated.

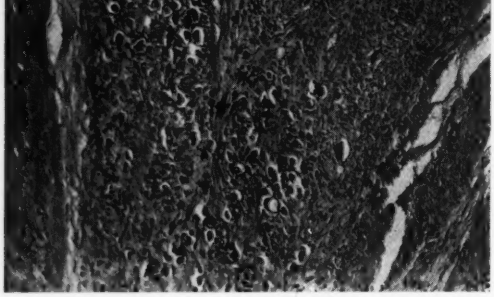

FIG. 85.—Tumour infiltration of muscle layer in rectum. (× 36.)

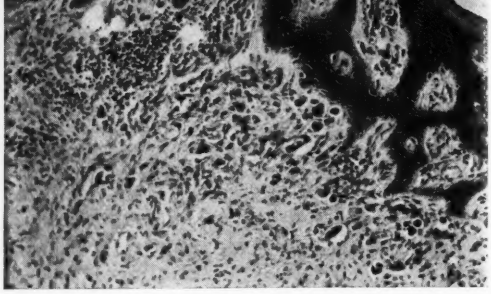

FIG. 86.—Malignant infiltration beneath squamous epithelium of perianal skin. (Post-mortem specimen.) (× 36.)

The perianal skin showed intense fibrosis in the dermis, in which were lying occasional groups of cells (*Fig.* 86.) These were mainly large, with hyperchromatic nuclei, but an occasional signet-ring form was discovered. A tendency to perineural distribution in the deeper layers was an additional indication, if any were needed, that these cells, were, in fact, carcinomatous. Immediately beneath the epidermis were accumulated numerous large cells of similar type. Scattered throughout the fibrous matrix occasional inflammatory cells were found, but eosinophils were not a feature.

To summarize, a female, aged 59 years, died in cachexia after fifteen months' history with symptoms predominately associated with the lower bowel, including persistent diarrhœa and vague lower abdominal pain. At post-mortem examination an anaplastic scirrhous carcinoma was found in the stomach, with generalized infiltration of the remainder of the bowel, together with involvement of the skin around the anus.

DISCUSSION

From the data presented there seems no doubt that this case is one of anaplastic carcinoma of the stomach of scirrhous type. In common with other observers, Dixon and Stevens (1936) and Saphir and Parker (1943), the findings in this case support the view that the condition is an anaplastic variety of adenocarcinoma with a tendency to mucin production by the individual cells and to fibrous overgrowth. The intense fibrosis has been stressed by Ewing (1940), who used the term 'sclerosing fibrocarcinoma' in the most advanced cases.

Certain features of the clinical history merit attention. The patient's symptoms and physical signs from the beginning until four months before death, when vomiting commenced, were always associated with the lower bowel. The most constant features were constipation and diarrhœa, together with pain in the right lower abdomen. In the early stages a diagnosis of carcinoma of the ascending colon was made with some confidence in view of the radiographic evidence (*Fig.* 76). Later the possibility of lymphogranuloma was seriously considered. It was not until two months before death, when radiographs showed pyloric obstruction, that attention was focused on the stomach (*Fig.* 80).

In reviewing the literature, complete absence of any signs pointing to the stomach as the site of origin of the disease is rare. Dixon and Stevens (1936) reported 2 of their cases as having symptoms mostly referable to the colon.

The interesting pathological features presented by this case are numerous. It seems reasonable to assume that the small area of thickening felt in the ascending colon at the time of the first operation, represented the commencement of one of the fibrous constrictions which became such a marked feature of the later stages of the disease, although no histological proof of this fact was obtained. It should be stressed that on examining the original sections of the operation specimen for the second time after the post-mortem findings were known, no evidence of malignancy could be demonstrated. It is possible that when the tissue was chosen from the large hemicolectomy specimen a small area of fibrous thickening was overlooked.

Spread of malignant disease through the bowel from carcinoma of the stomach has been recorded by many observers. An early account by Welch (1885) suggested that spread occurred by " implantation of cancerous elements from the primary into the intestines ". This view was held for some time, but is now considered most unlikely. Kaufmann (1929) considered " the assumption of submucous dissemination within the innumerable lymph-channels " was more logical. This diffuse invasion of the intestine in which lymphatic permeation seems to play a principal role is rare. The most frequent type of intestinal invasion occurs in cases of peritoneal carcinosis, where the mesentery and serous surface of the bowel contain tumour deposits. Secondary infiltration of the bowel wall occurs, with penetration of the muscularis and subsequent malignant ulceration of the mucosa in some cases. Good accounts of this latter phenomenon are given by Toyosumi (1908), Lyons (1924), and Willis (1934).

In the case presented here diffuse lymphatic spread within the wall of the bowel was clearly

evidenced microscopically by the plugging of the majority of the small submucous lymphatics in the stomach, duodenum, and jejunum (*Fig.* 84). Further along the bowel plugging and permeation of both submucous and subserous lymphatics was a marked feature. Evidently lymph-node and plexus blockage and retrograde spread had occurred. This diffuse lymphatic plugging was doubtless the cause of the terminal ascites.

The spread into the anal canal and even beyond it to the skin surrounding the anal orifice is most unusual, but here again evidence of lymphatic permeation, particularly around small nerves, was a feature of the microscopical appearances.

Willis (1948) has pointed out that widespread permeation does not necessarily proceed solely from the primary growth, but also from many embolic metastatic foci in lymph-nodes and plexuses, from which further permeation occurs, thus linking numerous areas.

The remarkable feature of this particular case is the very extensive invasion of the bowel, with complete absence of demonstrable invasion or metastasis beyond the gastro-intestinal system, its peritoneum, and lymphatic plexuses. Although the perianal skin can hardly be termed part of the gastro-intestinal system, yet the spread into it has evidently occurred by direct lymphatic permeation.

A question of terminology in association with the spread of this tumour is perhaps worthy of brief mention. Willis (1934) has stressed the abuse of the term ' metastasis ' in many cases when, in fact, direct extension of tumour has occurred. Thus, in the literature of so-called ' metastases ' of the intestines, the majority of cases are examples of direct spread. Willis (1931) has pointed out how very rare true intestinal metastases are.

In the case here described, much of the extension of the tumour is by direct spread, but if the theory of lymphatic-node or plexus blockage with subsequent permeation is to be accepted, then a certain degree of true metastatic spread has also taken place, and the changes might be properly described as carcinoma of the stomach spreading directly by submucous lymphatic permeation and metastasizing to local serous lymph-nodes and lymph-plexuses, from which further direct spread by permeation has taken place.

The exact designation of carcinoma type in this case gives rise to some difficulty. The stomach tumour, particularly in the pyloric region, suggests a colloid type, whereas the intense fibrosis both in the stomach, but more particularly in the rest of the bowel, seems to suggest ' linitis plastica ' as a more apt title. Ewing's ' diffuse scirrhous carcinoma ' seems the best description, but this case certainly upholds the dictum of Stout (1943), repeated by Willis (1948), that there is no value in attaching " histologically descriptive adjectives and prefixes to gastric carcinoma ".

SUMMARY

1. A case of diffuse scirrhous carcinoma of the stomach with spread exclusively to the remainder of the bowel and perianal skin is described, together with a brief review of the literature.

2. The unusual clinical history is noted.

3. The findings of carcinomatous spread to the perianal skin is described.

4. The method of tumour spread is discussed.

My thanks are due to Mr. E. Stanley Lee, Surgeon to the Westminster Hospital, for permission to make use of the clinical notes of this case and for many helpful suggestions ; and to Dr. Peter Hansell, of the Photographic Department of the Hospital.

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PULMONARY EMBOLISM ARISING FROM THE GREAT SAPHENOUS VEIN

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VENOUS thrombosis of the lower limbs has been divided by Oschner and de Bakey (1939) into two distinct types ; phlebothrombosis (acute thrombosis or deep thrombosis) in which the intravascular clot of blood is not associated with inflammation of the vein wall, and thrombophlebitis (chronic thrombosis or superficial thrombosis) in which inflammation of the intima precedes the formation of the thrombus. From the pathology of phlebothrombosis it is obvious, since the vessel wall is not involved and there is no attachment of the clot, that embolic phenomena are likely to be common ; in thrombophlebitis, since the clot is adherent to the inflamed vessel wall, there is little tendency for embolic phenomena to occur (Oschner and de Bakey, 1939).

Mayo Clinic workers argue that there is some association between the two types (Allen, Barker, and Hines, 1946). This is almost certainly true.

In the case of phlebothrombosis, the vein wall is irritated by the clot lying within it and undergoes an inflammation, with consequent adherence of the clot. Phlebothrombosis thus proceeds to thrombophlebitis as a step in its resolution, and once thrombophlebitis is established, the danger of embolism is usually considered to be over. In thrombophlebitis treated by chemotherapy and rest in bed, immobility leads to slowing of the blood in the deep veins and phlebothrombosis may follow.

Phlebothrombosis and consequent embolism is essentially an affection of the deep veins. The superficial veins are not commonly affected by phlebothrombosis, and when they are the seat of thrombophlebitis, which they very often are, the intravenous clot is tightly adherent from the time of its formation and the liberation of emboli from it must be exceedingly rare; when embolism occurs in the course of superficial thrombophlebitis it cannot be assumed that the embolism has been liberated from the superficial vein unless it is shown that the deep veins are free of non-adherent clot; the embolus might well have formed in a coincident phlebothrombosis of the deep vein. Indeed this origin of the embolus would best suit current views of venous thrombosis.

Recently we have traced a pulmonary embolism to the great saphenous vein in a patient suffering from thrombophlebitis of that vein. We report the case as one of proved embolism from a superficial thrombophlebitis and we feel that it lends weight to the arguments of those who advise the ligation and division of the saphenous vein in the treatment of superficial thrombophlebitis.

CASE REPORT

B. C., a married woman aged 62 years, was admitted to a surgical ward of this hospital on Jan. 20, 1948, complaining of pain of one week's duration in her right leg and thigh. For as long as she could remember she had had large veins in her legs. One week before admission the veins in the right leg had become red, hard, tender, and painful, and had remained so in spite of rest at home.

ON EXAMINATION.—The right great saphenous vein in the upper two-thirds of the leg and lower third of the thigh was thrombosed, inflamed, and tender, and the skin over it was red, shiny, and adherent. A second area in the proximal third of the same vein was similarly affected. Clinical tests for patency of the deep veins were inconclusive; there was no tenderness along the midline of the calf or instep and dorsiflexion of the foot produced no pain in the calf, though extreme dorsiflexion seemed to stretch painfully the inflamed saphenous vein. The small saphenous vein was not involved. The veins on the left side were varicose, but not inflamed or thrombosed. The lungs showed no abnormal clinical signs. The heart-sounds were distant and a soft systolic bruit at the apex had all the characteristics of a functional murmur. The blood-pressure was 160/86, the temperature 99·6°, the pulse-rate 96 per minute, and the respiratory rate 22 per minute.

TREATMENT.—The patient was put to bed and kept there, and 20,000 units of penicillin were given three-hourly. Progress was satisfactory until 9.30 p.m. on Jan. 28, when there was sudden faintness and a severe pain in the right chest. A diagnosis of pulmonary embolism was made and, in spite of the rarity of dislodgement of an embolus from a superficial vein, it was concluded that here the embolus had arisen from stagnation of blood in the great saphenous vein proximal to the area of thrombophlebitis. The absence of œdema, colour changes, and calf tenderness, associated with a negative Homans' sign, supported this diagnosis. A heparin response test (de Takats, 1943) showing a pre-heparin coagulation time of 10 minutes, which, rising to 14½ minutes ten minutes after giving 1000 Toronto units (10 mg.) of heparin intravenously, indicated that the blood was not preternaturally liable to clot and further supported the belief that the embolus arose from the great saphenous vein rather than from deep veins coincidentally the seat of phlebothrombosis. Five thousand Toronto units (50 mg.) of heparin were given three-hourly intravenously for three doses and the drug was then discontinued to allow the coagulation time to return to normal before operation for ligation and division of the great saphenous vein at the groin.

At 10.30 a.m. on Jan. 29, the great saphenous vein was exposed under local anæsthetic. A free clot was found in a varix of the proximal end of the vein 2·5 cm. from its junction with the femoral vein. The clot showed a rough proximal surface which suggested that a portion had become detached (*Fig.* 87). The wall of the vein

FIG. 87.—Diagrammatic section through varix showing clot and detached portion (dotted line).

appeared smooth and normal. The great saphenous vein was doubly ligated, at its junction with the femoral vein above the clot and also 2·5 cm. below the varix and clot. A medial tributary, joining the main trunk immediately proximal to the varix was also ligated and divided (*Fig.* 88) and the clot-containing varicose segment of

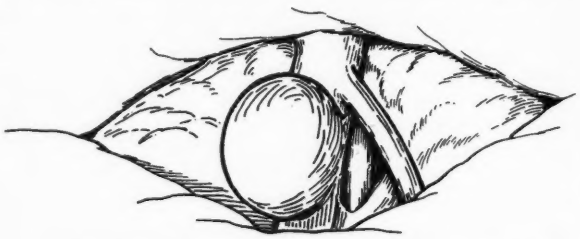

FIG. 88.—Great saphenous vein exposed at operation, showing varix and medial tributary. Femoral vein exposed in angle between two superficial veins.

vein was then removed. The femoral vein was explored and no abnormality was detected in it. The incision was closed and the patient returned to bed.

In order to prove that the embolus did arise from the superficial vein, a venogram was performed at 4 p.m. on the same day. The injection was carried out through a superficial vein on the dorsum of the right foot, without a tourniquet. This showed patency of the small saphenous and deep veins of the leg and thigh, and the ready drainage of the blood from the superficial to the deep venous system pointed to complete patency of the deep veins (*Fig.* 89).

Radiography of the lungs showed quite a large area of infarction of the right lung.

PROGRESS.—The patient was allowed up on the day following operation and was discharged from the ward on Feb. 11 with slight stiffness of the right leg and some œdema of the foot and ankle towards the end of the day, the œdema disappearing at night.

On Feb. 20 the patient looked fit and well and was doing a full day's work as a housewife. There was some œdema still in the foot at the end of the day, but the

swelling was substantially less than it had been. On March 16 the patient had no complaints.

PATHOLOGICAL REPORT.—No abnormality or infection of vein. Thrombus consists of recent blood-clot showing no evidence of organization.

DISCUSSION

A normal sensitivity of the coagulation mechanism to heparin strongly suggested that there was not a general tendency to spontaneous intravascular clotting; at least the heparin test is usually so interpreted.

A B

FIG. 89.—Venograms of deep veins, showing complete patency. A, Femoral vein; B, Deep leg veins.

This, together with the absence of clinical signs of phlebothrombosis of the deep veins, led to the pre-operative diagnosis of superficial thrombophlebitis of the saphenous vein uncomplicated by deeper clot. It was considered probable that the embolus had arisen from unorganized clot deposited proximal to the phlebitic area as a result of the slowing of the venous stream in the vein proximal to the primary clot.

The unorganized and recent character of the clot found, no less than the operative and venographic demonstration of the patency of the deep veins, confirmed the pre-operative assumption of the superficial origin of the embolus, which was further supported by the non-recurrence of embolism even though the patient was treated by early ambulation and without heparin.

In a review of the literature we have been unable to determine how frequently embolism has complicated thrombophlebitis. This difficulty, appreciated by other investigators, is due to two causes: (1) In the early literature the term 'thrombophlebitis' included cases of phlebothrombosis, which was not separately distinguished; and (2) in reported cases of an undoubted association between thrombophlebitis and embolism the possibility of associated phlebothrombosis of the deep veins has not been definitely excluded.

In thrombophlebitis there are two possible sites from which an embolus may arise. In the superficial vein phlebothrombosis may occur in the proximal segment, laid down on the proximal end of the primary clot, or separate from and proximal to that clot. This source is rendered harmless by early ligation and division of the vein at the groin. Bed-rest favours the development of phlebothrombosis in the deep veins; this embolus-producing complication is prevented by early ambulation, which can be permitted safely only if it is preceded by high saphenous ligation. It would appear to us that these advantages justify the employment of early ligation and division of the superficial vein at the groin in the treatment of saphenous thrombophlebitis, for it cannot now be assumed that superficial thrombophlebitis is free from the risk of pulmonary embolism. We may add that when we have performed ligation and division, the pain of the thrombophlebitis has been relieved.

SUMMARY

The pathology of phlebothrombosis and thrombophlebitis is briefly discussed.

A case of pulmonary embolism arising from an area of phlebothrombosis in the great saphenous vein is described. The phlebothrombosis developed in a normal segment of vein proximal to an area of pre-existent thrombophlebitis, and its clot was not in direct continuity with the fixed clot of the thrombophlebitis. It is concluded therefore that the phlebothrombosis occurred, not as an extension of the thrombotic process below, but as a new and separate coagulative process, probably the result of lessened rapidity of flow in the blind segment of vein above the primary clot.

The advantages of division and early ambulation in the treatment of thrombophlebitis are outlined.

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PRIMARY CARCINOMA OF THE TRACHEA

By J. F. PANTRIDGE, Belfast

Primary carcinoma of the trachea is of sufficient rarity to warrant the recording of two cases that have appeared in the autopsy material of this Hospital during the past year.

CASE REPORTS

Case 1.—A 52-year-old man gave a history of exposure to ' gas ' during the first world war. He had complained of persistent cough with tenacious sputum for four years before coming to hospital. Blood-stained sputum was noted one week before admission. He also complained of dysphagia and of persistent pain in the region of the right sternoclavicular joint.

Fig. 90.—*Case* 1. Trachea, showing site of lesion.

Physical examination showed him to be cyanosed and orthopnœic, with some inspiratory stridor. The finger-nails were clubbed. Enlarged glands were palpable in the anterior and posterior triangle on both sides of the neck.

Increasing respiratory embarrassment necessitated tracheotomy. One week after this operation his condition suddenly deteriorated and signs of bilateral bronchopneumonia appeared. He died two months after admission to hospital.

Post-mortem Examination.—This confirmed the presence of finger clubbing and enlargement of the cervical glands. The margins of the tracheotomy opening showed a mild infection. On opening the larynx and trachea tumour tissue was seen to involve the lower part of the larynx to within half an inch of the vocal cords. The growth involved all coats of the trachea from its commencement to the level of the fourth tracheal ring. From the fourth to the seventh tracheal rings the growth was confined to the mucosa and submucosa (*Fig.* 90). There was extensive invasion of the peritracheal tissues. The thyroid and the majority of the cervical glands were completely replaced by growth. There was invasion of the medial end of the right clavicle and right sternoclavicular joint.

The trachea below the level of the tumour and the bronchi showed an acute inflammatory change. The lungs showed a bilateral confluent bronchopneumonia, which had in some areas progressed to abscess formation. A few nodules of firm white tumour tissue were found in the right lobe of the liver.

The tracheal tumour and the deposits in the cervical glands, thyroid, and liver presented similar histological

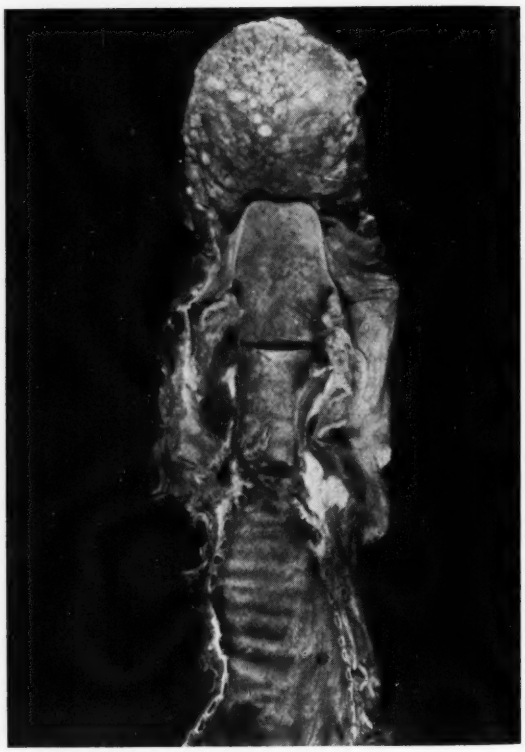

Fig. 91.—*Case* 2. Site of tumour in trachea

features. The appearance was that of a highly cellular tumour composed of round or oval cells with scanty cytoplasm and deeply-staining nuclei. The cells were commonly irregularly arranged, but in some sections they were seen to grow in cords. The stroma was scanty, but in some areas shows a branching fir-tree arrangement.

The histological characteristics were those of an oat-cell carcinoma, identical with that of bronchial origin.

Case 2.—The patient, a male aged 69, was admitted to hospital in a moribund condition, deeply cyanosed, and with stertorous breathing. He was unable to give a coherent history and died immediately after admission.

Post-mortem Examination.—The anterior and lateral walls of the trachea from its second to fourth rings were the seat of a tumour (*Fig.* 91). The growth in the anterior wall had destroyed the mucous membrane,

with the formation of a shallow ulcer. On the right side the growth extended through the wall of the trachea into the peritracheal tissues. In this region the cartilage rings of the trachea were softened and interrupted.

The trachea below the level of the growth, and the bronchi, were acutely congested. Patchy consolidation was evident in the lower lobes of both lungs.

Histological examination showed the tumour to be an anaplastic squamous carcinoma. Large sinusoidal vessels, some thrombosed, were found in relation to the area of ulceration. Deposits of tumour were seen throughout the wall of the trachea at the level of the growth. The peritracheal lymphatics were invaded.

The lungs were the seat of a bronchopneumonia.

An incidental finding at post-mortem was a hæmangioma on the pulmonary valve.

DISCUSSION

The trachea is comparatively infrequently the site of a primary carcinoma. D'Aunoy and Zoeller (1931) note that the statistics of the Pathological Institute of Basle showed, out of a total of 1078 malignant growths, 1 tracheal and 9 laryngeal involvements.

These authors also note that at the Charity Hospital of New Orleans over a period of twenty-three years (1906–29) 68 laryngeal new growths and 1 tracheal tumour were observed.

Culp (1938) found only 2 cases of primary carcinoma of the trachea in a survey of 47,600 autopsies.

The total number of cases of tracheal carcinoma reported is also small. Thus Von Bruns (1898) was able to collect only 31 cases from the literature. Additional cases have been collected by Krieg (1908), Lombard and Baldenweck (1914), and D'Aunoy and Zoeller (1931). An extensive review by Culp (1938) indicated that the total number of recorded cases in 1936 was 147.

There is general agreement in the literature that primary carcinoma of the trachea is more common in males; 63 per cent of the cases reviewed by Culp were in men.

The recorded cases show a fairly even distribution among the age periods between 30 and 70 years.

Culp and D'Aunoy and Zoeller are in agreement that tracheal carcinoma most commonly arose in the lower one-third of the trachea at or near the bifurcation.

Most reports show that the posterior wall is more commonly involved than the anterior wall.

Histologically the tumours have been squamous-cell carcinoma, adenocarcinoma, or of an undifferentiated (including oat-cell) type. The cell types encountered in the tracheal tumours are, therefore, similar to those found in carcinoma of bronchial origin. It is of interest that a relatively higher proportion of tracheal tumours are squamous-cell in type. Culp found that squamous carcinoma was the commonest type of carcinoma arising from the trachea, constituting 35 per cent of tumours classified histologically. In contrast Willis (1948) noted squamous carcinoma was the least common type of carcinoma arising from the bronchi. A squamous carcinoma was found in only 27 per cent of his cases of primary carcinoma of the lung.

The review of Culp indicates that the commonest type of tracheal carcinoma to metastasize is the squamous-cell variety.

In the cases reported here the squamous-cell tumour showed local extension only, whereas the oat-cell carcinoma showed wide-spread metastases.

SUMMARY

Two cases of primary carcinoma of the trachea are reported, one a squamous-cell carcinoma, the other an oat-cell tumour.

Both tumours arose from the upper third of the trachea.

A brief review of the literature indicates the infrequent occurrence of primary tracheal carcinoma.

I am indebted to Professor J. H. Biggart, C.B.E., for help in the preparation of these case reports.

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FAT NECROSIS OF THE BREAST

WITH REPORT OF A CASE IN A MALE

BY MAURICE SILVERSTONE, LIVERPOOL

FAT necrosis of the breast is an uncommon lesion. Lee and Adair (1924) considered its frequency relative to carcinoma of the breast to be about 2½ per cent. Menville (1935) found 58 fatty-tissue tumours amongst 3000 breast tumours. Of these, there were 25 cases of fat necrosis, 9 cases of secondary xanthomatous degeneration which were probably secondary to fat necrosis, as well as 24 lipomata. Though cases had been recorded previously—Hebb (1890), Targett (1896)—it was not till the publication of the articles by Lee and Adair (1920, 1922) that the importance of the condition, because of its close clinical resemblance to carcinoma, was generally recognized.

AETIOLOGY

This is not known, and for that reason extra-peritoneal fat necrosis has been variously termed 'saponifying necrosis' (Shattock, 1896), 'traumatic fat necrosis' (Lanz, 1898; Lee and Adair, 1920), 'mammary panniculitis' (Cookson, 1930), and 'lipogranulomatosis' (Harbitz, 1935). Clinically it has been found to follow trauma—massive enough to have produced ecchymosis, or mild and remote,

even ten years before the discovery of the lesion. Lee and Adair (1920, 1922) found a history of trauma in all their 6 cases, but Binkley (1939) found a history of trauma in only 50 per cent of cases collected in their clinic since the original report. It has followed the application of an ice bag (Cohen, 1923), after subcutaneous infusions (Levin, 1931), and following radical mastectomy, where it has closely simulated a recurrence. Outside the breast area it has occurred after hypodermic injections, has been seen in the newborn, where it has probably been caused by trauma (Fox, 1933), in the faces of children exposed to cold (Haxthausen, 1940), and has been found in the scrotum of boys around puberty—three cases being recorded by Hinman and Johnson (1939). The condition of ' relapsing febrile nodular non-suppurative panniculitis ', described by Weber (1925) and Christian (1928), the Weber-Christian syndrome, is probably allied to this. The syndrome is thought to be due to a low-grade systemic infection, though Weber (1935) considered the condition might also be produced in idiosyncratic individuals by the exhibition of iodides and bromides.

Experimentally, non-pancreatic fat necrosis has been produced by intraperitoneal injection of lipase by Neal and Ellis (1930), and Farr (1923) produced fat necrosis in the pig by subcutaneous pressure with forceps. He thought fat necrosis was due to simple pressure necrosis from anæmia, with secondary fat splitting. Because he found that fat splitting would occur in the presence of 10 per cent formol, he did not think it necessary to postulate lipase action, though a lipase was present in body tissues as well as blood. He stressed the difference between fat splitting and fat necrosis, considering the necrosis to be the principal event, and the saponification an accompaniment. Gottesman and Zemansky (1927) suggested that the escape of fatty material from a breast duct or a cystic mastitic space into the tissues and its subsequent decomposition might act as the irritant which produced necrosis of the adjacent fat. In their series of 20 cases trauma was an infrequent factor. Hadfield (1930) considered the central feature of the process to be a slow aseptic saponification of neutral fat by blood or tissue lipase, analogous to pancreatic fat necrosis but much milder in degree and independent of any direct action of pancreatic lipase.

It would seem that fat necrosis is due to a local disturbance of lipoid metabolism produced by secondary factors, e.g., trauma, pressure ischæmia—the weight of a pendulous breast—infection, and so on ; and it is likely that in addition some endogenous factor exists. This may be, for example, a more abundant or more easily activated lipase.

PATHOLOGY

Gottesman and Zemansky (1927) describe four groups :—

1. Fat necrosis occurring in fat overlying a breast.

2. Fat necrosis occurring as a single tumour in an otherwise normal breast.

3. Fat necrosis occurring as multiple punctate areas in a breast the seat of some other pathology—usually cystic mastitis.

(Hadfield, 1930, does not accept this group as a true fat necrosis, because the tissue reaction, though similar, is secondary to duct obstruction, and the fat is derived from the secretion of the gland.)

4. Fat necrosis occurring in a lipoma of the breast.

The macroscopical appearance of the lesion has been described by Hadfield (1930). The lesion is usually well defined, but in its early and late stages may be diffuse. The early lesion may occupy one or two fat lobules, and may be $\frac{1}{2} \times \frac{1}{4}$ cm. to 4×2 cm. It is opaque, white, and dull, the periphery of the lesion being pinkish. Central liquefaction may be seen even in an early lesion, with the production of a pool of yellowish, oily fluid. The area becomes more chalky, and as it is invaded by peripheral fibrous tissue the unbroken central chalky area is split up, the edge becoming irregular, and the lobular shape is lost. At this stage the appearance is that of many minute, rounded and irregular fragments rather like candle grease lying in pinkish connective tissue. Often some central liquefaction occurs, with the production of a pseudocyst. From this stage the appearance is altered by the slow development of a fibrous-tissue capsule, by cicatrization, and more extensive cyst formation, but small fragments of white or whitish-yellow necrotic fat resembling candle fat remains for several months or more.

At all stages, the lesion is firm to the touch, but not as hard as carcinoma. On section, the areas of white or yellow opacity are larger and more irregular in shape, and do not radiate from the centre as do the fatty streaks in carcinoma. There is no concavity when cut across, nor is there the rich development of pink translucent bands of fibrous tissue such as is seen in carcinoma.

In some cases associated with trauma, there may be pigmentation from hæmosiderin, and in old cases there is irregular calcification and the lesion is usually cystic. The microscopical picture has been well described by Stewart (1924), who pointed out that it differed from the fat necrosis in pancreatitis in that phagocytosis of fat-cell disintegration and fibroblastic proliferation keep pace with cell destruction. The lesion is characterized by infiltration of mononuclear phagocytes, many of which become filled with particles of necrotic fat. These are the so-called foam cells, which may be mistaken for embryonal fat cells (F. Parkes Weber, 1925). In addition there may be a heavy cellular infiltration of lymphocytes, plasma cells, occasional polymorphonuclear cells, and in later stages giant cells are seen (Dunphy, 1939).

CLINICAL FEATURES

The cases described hitherto in the literature readily available have all occurred in women. They have been seen in women between 19 and 60, though the commonest age incidence is 40 to 50. The breasts are usually pendulous and heavy, and the patients obese, though cases have been recorded in thin patients with emaciated breasts. The condition in the absence of trauma occurs more frequently in the left breast and in the upper and outer quadrant. The length of history may be two years to ten days. A history of trauma is obtained in 40 per cent of cases. The lesion may increase in size and be associated with some pain and tenderness. It

presents as a firm, well-defined area, is attached to skin in 50 per cent of cases, and may be associated with *peau d'orange*. In about 10 per cent of cases there is retraction of the nipple, and in 10 per cent of cases the lesion is fixed deeply. There are no palpable axillary glands. A diagnosis of carcinoma had been made in 28 per cent of the cases collected by Menville (1935).

TREATMENT

The majority of cases reported have been treated by excision of the area. Hadfield (1930) found that 26 per cent of the cases had had a radical mastectomy performed under the mistaken diagnosis of carcinoma—in some cases even after local excision and inspection of the tumour.

Rowntree (1925) thought the lesion, if left, would resolve. Dalgaard (1945), in recording 3 cases, described a patient with a lesion in each breast. Local excision was performed on one side and the diagnosis was confirmed histologically. The lesion in the other breast was left, and when the patient was seen five years later the breast was normal.

It is evident, however, that in the breast, where fat necrosis simulates carcinoma so closely and occurs in the same age group, local excision and histological examination must be carried out.

CASE REPORT

The patient, a married man aged 51, an engineer's representative by occupation, was first seen on April 28, 1947. He complained of a swelling under his left arm of two weeks' duration, and of a more recent swelling—of two days' duration—in the region of his right costal margin.

For the week before he noted the swelling under the arm, he had noticed a sensation of pressure over his heart when he lay on his left or right side, and found it more comfortable to sleep on his back. He noticed the swelling under the arm first. Four days later the swelling became red; it was hard and not painful. He thought it had remained stationary. The swelling over his costal margin was also painless, and had not increased in size since he first noticed it. He had no other symptoms and was otherwise quite well. There was no history of trauma. His past history included a gunshot wound of the head in 1916 and a history of dermatitis. The latter had first occurred while he was working at an ordnance factory, and was said to be due to the irritating effect of 'C.E.' powder. It was associated with intense irritation, and the sensation of burning of the exposed skin, with swelling of the face, puffiness of the eyes, and the presence of pimply sores. It induced scratching and was associated with general irritability. In 1944 he had a severe breakdown in health, his face and hands were covered with spots and sores, and he was confined to bed for two months. He left the factory in January, 1945, and had had no recurrence since leaving.

On Examination.—He was a well-covered plumpish man, without being obese. There was a swelling in the upper and outer quadrant of the left breast, which was hard, had a somewhat ill-defined edge, and was adherent to skin, but not deeply. It was about 2½ cm. in diameter. There were no palpable axillary or supraclavicular glands. Immediately below the right costal margin was another swelling, slightly oval, hard, but again fading rather into the neighbouring structures. It was 1½ cm. in diameter, fixed to the lower edge of the rib, but not definitely attached to skin. In the absence of this second swelling a diagnosis of carcinoma of the breast would have confidently been made.

On Admission (May 7).—The swellings previously noted were about the same size or possibly a little larger. The patient had, four days before admission, noticed a further swelling, this time at his umbilicus. This swelling was associated with aching pain, which was relieved by wearing a broad belt. He thought it was increasing in size. The swelling was deep to, and below, the umbilicus, it was hard, had a slightly irregular surface, was fixed deeply but not to the overlying skin, which was normal in appearance. Clinical examination showed no abnormality in any system; there was no evidence of any focal sepsis. Radiography of his chest was normal, as was a complete examination of his blood.

At Operation (May 9).—The swelling together with the adjacent tissue was excised from the breast. Macroscopically the lesion was grey, with some whitish areas, was not concave on section, and contained no cysts. Histological examination revealed follicular formations in the fatty tissue—the structure being a giant-cell system, surrounded by mononuclear infiltration admixed with polymorphs. The follicles also contained large foamy cells. The section suggested traumatic fatty necrosis. The lesion at the costal margin and the one at the umbilicus were then also excised. Histological examination revealed a "chronic granulomatous condition of the type seen in fat necrosis" (Professor Sheehan).

On May 24 the patient noted a small tender swelling below his right ear.

He stated that this was how the other swellings had started. When seen two months later, all the scars had healed, there was no swelling below his ear, and no further swellings had appeared.

COMMENT

It is likely that this is the first case of fat necrosis in a male on record. It completes the analogy between the male and female breasts in the diseases to which they are liable. Unfortunately, it does not help in the elucidation of the condition. Trauma was not a factor in this case, and pressure necrosis from the weight of a dependent breast was obviously not in question. The history of dermatitis in 1944 with the possible trauma from scratching seems too remote, and, moreover, the dermatitis occurred on the exposed areas. Unlike the majority of cases of fat necrosis, which are single, in this case there were a number of foci occurring consecutively. In this there is a resemblance to the Weber-Christian syndrome of relapsing febrile nodular non-suppurative panniculitis.

It would seem to show that fat necrosis occurring in the Weber-Christian syndrome is an incidental condition, the cause of which is not clear. The development of the fat necrosis must have some factor other than infection, in view of its rare occurrence.

It is important to realize, however, that fat necrosis can occur in conditions in which there is generalized sepsis, as otherwise the development of consecutive multiple foci may suggest a pyæmic condition.

I am indebted to Professor Sheehan and his Department for the histological report.

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A RARE CASE OF BONE DYSTROPHY

BY JOHN HALLIDAY, SYDNEY

CASE REPORT

HISTORY.—G. S. a girl aged 9 years, was referred to the Medical Out-Patient Department of Lewisham Hospital, Sydney, in January, 1933, by Dr. Cedric Cohen, Ophthalmic Surgeon to the Hospital. The child's mother stated that during the preceding few months she had become almost totally blind; failing vision had been present since 1931. The first series of X-ray examinations were made at this time (*see* Dr. Sear's report *below*).

FAMILY HISTORY.—The patient's father and mother were first cousins (father's mother and mother's mother being sisters). She was the fourth child in a family of five :—

1. Female, alive, aged 20 years, healthy.
2. Female, died, aged 16 months, "inward convulsions".
3. Female, died, aged 5½ years. Records from Royal Alexandra Hospital for Children show that she died from acute encephalitis and bronchopneumonia. There is no mention of any bony abnormality.
4. The patient.
5. Male, alive, aged 2 years, healthy. No radiological abnormality of skull.

On the mother's side no familial disease was known: no history of father's family was obtainable.

FIG. 92.—Child at 3 years of age—suggesting that changes were already making their appearance.

FIG. 93.—Child at 6 years; swelling around the nose and thickening of the jaws.

The mother stated that the birth of this child had been a very prolonged labour, with instrumental delivery. Birth-weight was 9½ lb.; early development was apparently normal. The patient was breast-fed for fifteen months, had four teeth present at eight months, crawled at nine months, and walked at twelve months. Some "nasal obstruction" developed at this time, and the tonsils and adenoids were removed. The girl had been able to say a few words prior to this operation, but afterwards only made unintelligible sounds. The deciduous teeth became carious early, and were lost by the time she was 3 years old. Eight permanent teeth erupted early, but did not

develop normally. The mother was emphatic that no "thickening of the jaws" was observed until the child was five years of age, and that the next change was swelling around the nose.* At 7 years the child could sew, knit, and iron her clothes. About this time she had her first sudden attack of unconsciousness, and as it was also realized that her vision was failing she was taken to the Royal Alexandra Hospital for Children, Sydney, where bilateral optic atrophy was found. The child was very sensitive about her disabilities.

When first seen by the writer in 1933 the main complaint was of severe headache, mainly in the occipital region, and increasing fatigue. She also had frequent "screaming attacks" in her sleep.

PHYSICAL EXAMINATION.—The photographs (*Figs.* 94, 95) give a good indication of her general appearance and configuration. The skin of the face had a curious slate-grey colour and was stretched tightly over the underlying bone. Constant lateral nystagmoid movements of the eyes were present, and saliva dribbled intermittently from the corners of the mouth, which could not be closed owing to the deformity of the jaws. The optic disks showed advanced optic atrophy. Nerve deafness was present. The remaining teeth exhibited mal-eruption and were grossly carious.

The Wassermann reaction was negative and the blood-picture normal in all respects.

The patient was kept under constant observation for the next fifteen months, during which time she had one further attack of unconsciousness lasting fifteen minutes. Her headaches became more constant, and were associated at times with vomiting. She became completely blind in the left eye.

In March, 1934, she was admitted to the Royal Prince Alfred Hospital, where Dr. F. S. Hansman, Honorary Biochemist to the Hospital, carried out a series of observations on her calcium and phosphorous metabolism, and a further series of radiographs were taken and reported on by Dr. H. R. Sear, Honorary Radiologist. Their reports are attached. (*See* pp. 54, 55.)

The child died suddenly in her sleep at home. An autopsy was performed at the Royal Prince Alfred Hospital by Dr. G. F. Davies, Pathologist to the Hospital (*see* p. 57).

FIG. 94.—Indicates the general appearance and configuration of the patient: mouth unable to be closed owing to deformity of jaws; mal-erupted teeth.

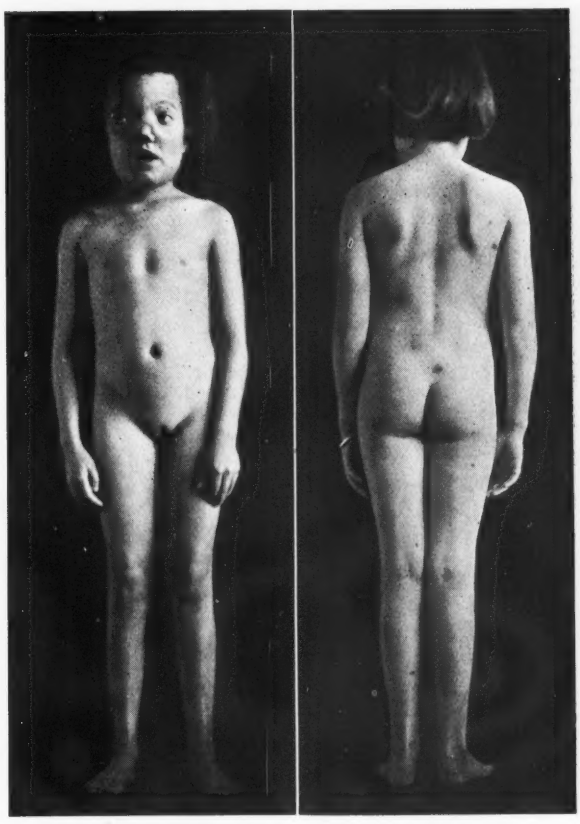

FIG. 95.—Child when about 10 years of age.

* A photograph taken at three years of age strongly suggested that changes were already present. (*Fig.* 92.)

X-RAY REPORTS

(*Dr. H. R. Sear*)

This child was first seen at the Royal Alexandra Hospital for Children, Sydney, in June, 1931, and later at the Royal Prince Alfred Hospital in March, 1934. The radiological examination on the first occasion showed the following features :—

FIG. 96.—Radiograph showing an extraordinary degree of thickening of the calvarium, especially the central parts of the frontal and parietal bones, and also at the base of the skull.

1. Skull (*Fig.* 96).—There was an extraordinary degree of thickening and sclerosis of the bones of the calvarium, most marked in the more central parts of the frontal and parietal bones, less prominent in the region of the coronal suture and in the posterior portion of the skull. The osteosclerosis and thickening were very pronounced at the base of the skull. The bones of the face, especially the maxillæ, were markedly involved ; there was enlargement of the bones by bony overgrowth accompanied by obliteration of the antral cavities ; the malar bones and mandible presented similar osteosclerosis and expansion, though not to such a marked degree.

FIG. 98.—Showing marked thinning of cortex of long bones.

2. Thorax and Shoulder Girdles (*Fig.* 97).—The ribs were widened and extremely dense, the condition being perhaps most marked in the mid-portion of the ribs. The clavicles were involved to an extraordinary degree, the thickening, expansion, and osteosclerosis being most marked in the mid-part and tapering away at the acromial ends, where osseous expansion and increased density were practically absent. The cervical vertebræ were very definitely involved, but the vertebræ below this region showed only slight changes.

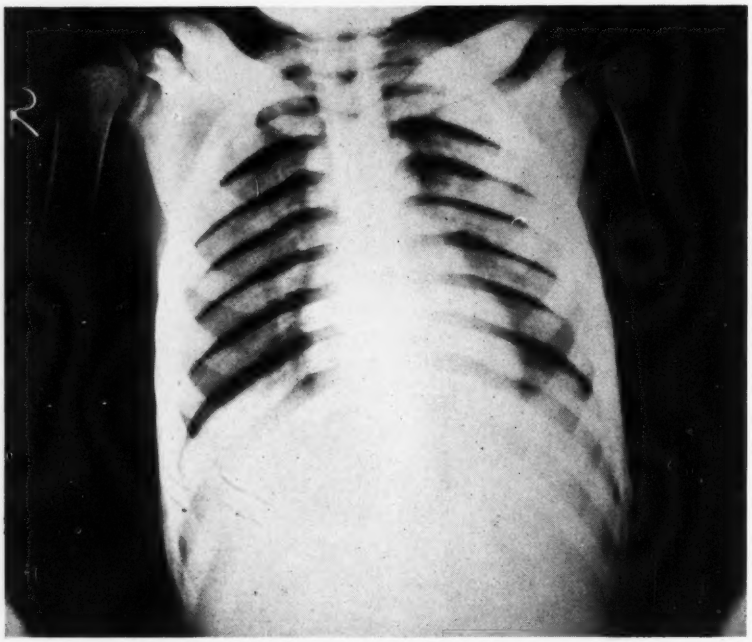

FIG. 97.—Radiograph of chest. Note widened and dense ribs ; also the expanded osteosclerotic clavicles and changes in cervical vertebræ ; also small nodule on inferior edge of 7th right rib 1½ in. from vertebral end.

3. Long Bones.—These presented a very unusual appearance—they were extraordinarily straight ; the cortex was thinner than usual (*Fig.* 98) and the calibre of the bones was unusually uniform throughout their length, being much wider than normal in the mid-part of the shafts. The trabeculation was slightly looser than usual.

Similar changes were present in the metacarpal (*Fig.* 99) and metatarsal bones (*Fig.* 100), and in the case of the former there appears to be actual ' ballooning ' out of the mid-part of the shafts. The cortex of the metacarpal and metatarsal bones was thinner than normal, except in the case of the 1st metacarpal, where there appears to be slight osteosclerosis. The phalanges show somewhat similar changes ; there was expansion of the shafts, which are unusually straight, and the trabeculation was looser in character.

The radiological diagnosis made at that time was ' atypical ' marble bones.

The child was subsequently admitted to the Royal Prince Alfred Hospital in 1934, and was X-rayed in March of that year. The same features were noted as three years before, although the condition had advanced in certain areas : thus the occipital bone was more thickened and sclerosed than at the previous examination, and the maxillæ appeared to be somewhat larger. Very little change had occurred in the condition of the clavicles and

FIG. 99.—Radiographs of forearms and hands, showing thinning of cortex of long bones, including metacarpals. Ballooning out of mid-parts of metacarpal shafts.

ribs. The long bones presented the same features as before, but the cortex had now slightly thickened in the case of the metacarpal bones.

A further X-ray examination was carried out later (April, 1934) when there appeared to be even more thickening in the bones of the calvarium, especially in the occipital region. The osteosclerosis and expansion had also advanced slightly in the maxillæ. The ribs and clavicles presented much the same features as before. The pelvis, which apparently had not been X-rayed before, showed osteosclerosis lateral to and just beneath the sacro-iliac synchondroses. No suggestion of ' marbling ' or osteosclerosis could be seen in the femora or tibiæ, but there was slightly more osteosclerosis in the upper ends of the radii. The cortex of the first metacarpal bones was slightly denser.

An interesting feature noted in the case of the ribs at the time of the first examination was the appearance of a small nodule on the inferior edge of the 7th right rib about 1½ in. from the vertebral end (*Fig.* 97). At the second examination in 1934 a similar small nodule had appeared on the inferior edge of the 6th left rib in a similar position ; later examination showed that these small nodules had increased slightly. The appearance suggests that they are due to some rupture of the periosteum in this region allowing a small outflow of osseous tissue at this point.

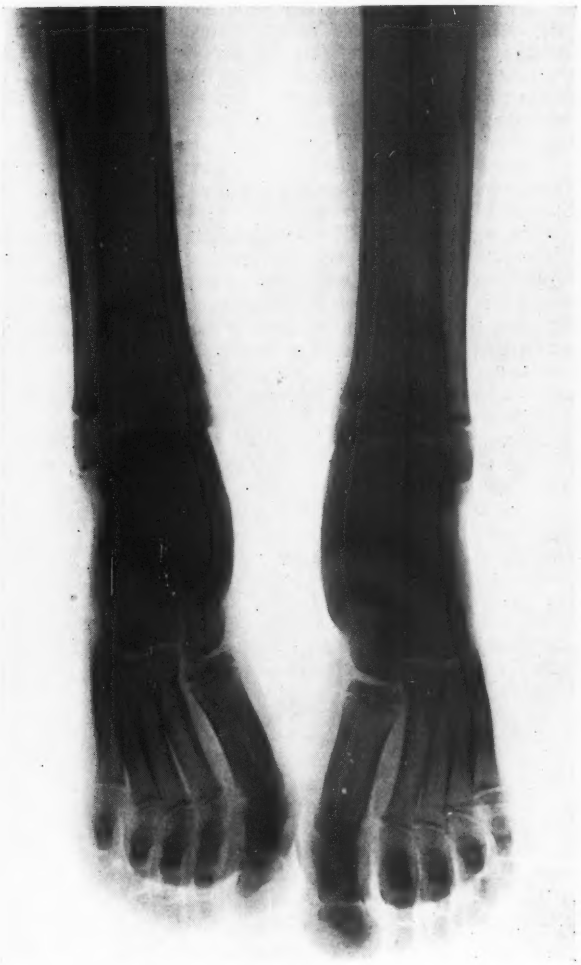

FIG. 100.—Shows thinning of cortex of long bones such as tibia and metatarsals : calibre of bones unusually uniform throughout their length.

Similar masses are seen in the periostitic type of leontiasis ossea, and also may occur in melorheostosis.

METABOLIC STUDY

(*Dr. F. S. Hansman*)

The patient was studied in the special metabolic ward of the Royal Prince Alfred Hospital, Sydney, from March 1 to April 6, 1934. There was a preliminary period of seven days for adjustment of diet and general observation.

The diet throughout the whole of the experimental period was one of 1800 calories. The first period began on March 8. There were two 4-day periods on diet alone, two 4-day periods during which 11 g. of calcium gluconate, equivalent to 1 g. of calcium, was administered by mouth daily, two periods of four days with the addition of 0·2 c.c. beryllium nitrate daily, and one period of four days with the addition of 0·5 c.c. beryllium nitrate daily. The results are shown in the table. The periods were marked off by carmine.

An analysis of the data gives the following results :—

		Calcium	*Phosphorus*	*Nitrogen*
Periods 1, 2	Intake per day	0·425	0·773	8·3
	Excretion per day	0·065	0·333	5·6
	Balance	+0·360	+0·440	+2·7
Periods 3, 4	Intake per day	1·428	0·761	8·3
	Excretion per day	0·445	0·349	5·7
	Balance	+0·982	+0·412	+2·6
Periods 5, 6, and 7	Intake per day	0·42	0·725	8·3
	Excretion per day	0·09	0·509	6·3
	Balance	+0·33	+0·216	+2·0

Calcium Metabolism.—There was an extraordinarily small excretion of calcium throughout the study. During the first two periods the total excretion of calcium per day was 0·065 g., indicating that the demand for calcium by the skeleton was very great, and that the katabolism of calcium was either remarkably small or that calcium liberated by the normal process of bone resorption is available for re-utilization in bone formation. The possibility of inadequate bone resorption will be discussed later.

During the eight days when an extra gramme of calcium was given daily, the retention, and apparently the utilization, of calcium was very great. The excretion of calcium in the urine remained at the same low level as in the first two periods, viz., in the vicinity of 0·02 g. per day. The fæcal calcium rose from 0·05 g. to 0·425 g. per day. One is not prepared to admit that this increase in the stool calcium represents unabsorbed calcium.

During the administration of beryllium the urinary calcium remained constant at 0·015 g. per day, and that in the stool at 0·077 g. per day.

The excretion of calcium was, therefore, apparently slightly raised when compared with the first two experimental periods, but the experiment cannot be considered clear-cut because the apparent increased excretion was due to a larger excretion during the first beryllium period which followed immediately the periods in which calcium gluconate was being given. Subsequent work on normal controls indicates that after the administration of large doses of calcium there is always a lag in the calcium excretion for at least twenty-four hours after the appearance of the carmine that marks the end of the experimental period. Probably the same phenomenon occurred in this case, so that the calcium excretion for the first beryllium period was increased by the previous administration of calcium. That this is probably the case is borne out by the fact that the increased dose of beryllium during the third period did not determine any additional increase in calcium elimination. The calcium excretion for this period was 0·05 g. per day, against 0·043 g. in the second beryllium period.

This patient had exceptional skeletal development, and the results of the experiments on the calcium and phosphorus balance indicate definitely that if calcium is required by the skeleton the whole of the calcium in the food is available and can be utilized; also that the irreducible minimal katabolic excretion of calcium is of an order significantly lower than that postulated by Aub or Sherman.

Phosphorus Metabolism.—The phosphorus balance remained constant for the first four periods, and it was not influenced by the administration of an extra gramme of calcium per day. The total excretion during the first two periods was 0·33 g. per day, and during the third and fourth periods 0·36 g. per day. It may be argued that the results obtained indicate a maximal retention of available phosphorus and that had more been available by increasing the intake of utilizable phosphorus, phosphorus would have been retained in proportion to the increased calcium retention. Be this as it may, there is evidence that the retention of calcium or phosphorus (*see below*) may be independent of each other.

The administration of beryllium determined an increased excretion of phosphorus, and this increase was solely due to an increased excretion of phosphorus in the urine. Now the phosphorus excreted in the urine is obtained predominantly from organic phosphorus (Peters and Van Slyke), the organic phosphorus being broken down by phosphates in the kidney, so that our results indicate that beryllium affected the renal function. The stool phosphorus remained the same as that of the first two periods (first two periods, excretion 0·163 g. per day; three beryllium periods, 0·158 g. per day). There is, therefore, no evidence that the beryllium acted by preventing phosphorus absorption, as postulated by Kay.

Though the phosphorus excretion during beryllium administration rose from 0·33 g. per day in the control periods, to 0·51 g. per day, the calcium excretion was unaffected, and this in spite of the fact that during the high-intake periods which immediately preceded the beryllium administration a great deal of calcium had been retained independent of phosphorus.

The independence of the calcium and phosphorus metabolism indicates that these elements can be retained and at least temporarily precipitated separately in the skeleton independently, i.e., $Ca_3(PO_4)_2$ and $CaCO_3$ can be precipitated separately; in other words, this is indirect evidence against the bone salt being a complex apatite salt.

Nitrogen Metabolism.—There was a constant retention of nitrogen at approximately the same level throughout the thirty-two days of the experiment. The positive balance was somewhat lower during the administration of beryllium, indicating the interdependence of nitrogen and phosphorus which has frequently been observed. That we were able to maintain a positive nitrogen balance indicates that the experimental diet was satisfactory. This is a tribute to the sister in charge of the metabolic ward, because the child was very difficult to handle on account of her blindness and deafness and the difficulty she had with mastication. She was very miserable for hours at a stretch, had a good deal of headache, and often cried for long periods, so that it required great tact to get her to eat constant amounts of food.

BLOOD EXAMINATIONS

Serum-calcium during first experimental period 9·5 mg. per cent.

Serum-phosphorus during first experimental period 5·0 mg. per cent.

Blood Morphology (*March* 22, 1934):—

Red blood-cells	5,130,000
Hæmoglobin value	98 per cent
Colour index	0·96
White blood-cells	6600

Differential Leucocyte Count: Neutrophils 59 per cent, Lymphocytes 38 per cent, Eosinophils 3 per cent.

Van den Bergh Test: Direct reaction negative; indirect reaction negative.

Blood Morphology (*April* 3, 1934):—

Red blood-cells	5,120,000
Hæmoglobin value	98 per cent
Colour index	0·96

Hæmatocrit reading (volume of packed red cells) = 44

Mean corpuscular hæmoglobin = 30·7

Mean hæmoglobin concentration = 34 per cent

Urinalysis showed no pathological constituents.

Discussion of Blood Findings.—The serum-calcium is somewhat low, and the serum-phosphorus normal for the child's age. The normal blood morphology is of extreme interest, showing that in spite of the great density of bone there was adequate normal hæmopoietic marrow present. This shows that resorption of bone, which plays such a large part in the normal configuration of the skeleton (Brash), was present in this case, otherwise there would have been no bone-marrow. That there should have been such a profound disturbance in the process of calcification and yet a normal hæmopoiesis suggests that these functions may be independent.

Beryllium was administered to see if the retention of calcium could be controlled. This element was used because of the results reported in rats by Kay.

The nitrate was the only salt available. We did not know the toxicity of this salt in the human being, so that the dose we gave may have been inadequate to obtain definite results.

POST-MORTEM EXAMINATION

(*Dr. G. F. Davies*)

The examination was performed about ten hours after the death on May 5, 1934 (Royal Prince Alfred Hospital, Sydney, Autopsy No. 6315). The body was that of a young girl aged 10 years. The trunk and limbs show development consistent with the age of the subject: there is no wasting or deformity of these parts. The head is very much enlarged.

Macroscopical Examination.—

Thyroid.—This is of normal size and shape and weighs 10·5 g. The cut surface has the normal dark-amber colour; there are no opaque areas of hyperplasia and no increase of fibrous tissue is visible.

Thymus.—The thymus can be easily separated from the surrounding areolar tissue; on section it has a bright pink colour and appears to contain no fat. It weighs 29 g. The right lobe is 7·8 cm. long, 3·0 cm. from side to side, and 1·5 cm. thick in its thickest part. The left lobe is 6·8 cm. long, 2·7 cm. wide, and 1·5 cm. thick.

Several enlarged *lymph-glands* are present in the mediastinum. One, which measures 1·6 cm. in its longest diameter, is situated immediately caudal to the left main bronchus and contains a focus of caseation, 6 mm. in diameter. Another lymph-gland, found caudal to the bifurcation of the trachea, is 3 cm. in diameter and contains a pale area which is 1·3 cm. across and appears to be partly caseous.

Lungs and Pleural Cavity.—No excess of fluid is found in either pleural cavity. Both lungs are normal except for slight terminal hypostatic congestion. There is no excess of fluid in the pericardial cavity.

Heart.—The subepicardial fat is not increased in amount, but there is slight opacity of the epicardium over the anterior aspect of the heart. The foramen ovale is not patent. A small Chiari network is present in the right atrium. Weight of heart 130 g.

There is nothing of note in *spleen*, *liver*, *kidney*, *œsophagus*, *stomach*, or any of the abdominal viscera.

Ovaries.—Both ovaries are enlarged and more globular than usual. The right ovary is 2·56 cm. from side to side, 1·37 cm. in an anteroposterior direction, and 1·67 cm. from the hilum to the convex surface. Similar measurements of the left ovary 2·35 cm., 1·28 cm., and 1·56 cm. respectively. On section both ovaries are seen to contain follicular cysts, which vary in diameter from 5 mm. to 9 mm.

Microscopical Examination.—

Thyroid Gland.—There is a pronounced and uniform interlobular and intralobular fibrosis, the fibrous tissue being dense and lowly cellular. Only very slight and focal epithelial hyperplasia is present.

Parathyroid Glands.—Chief and oxyphil cells are both present. Though the former predominate, the latter are found in considerable numbers and in some areas are equal in number to the chief cells. There are also many groups of cells with cytoplasm which is eosinophilic, slightly granular, and small in amount, so that the nuclei appear closely packed together.

Thymus.—This shows no unusual features. The Hassall's corpuscles contain a variable amount of keratin. Some are large and almost completely keratinized, while others are smaller and consist entirely of squame cells. Some are cystic and others have deposits of calcium in the degenerate central cells, while a few are completely calcified. Only a few small groups of fat cells are present in the area sectioned.

Lymph-glands.—A lymph-gland caudal to the bifurcation of the trachea and one caudal to the left bronchus both contain large areas of tuberculous caseation surrounded by zones of fibrous tissue. There are clusters of tubercle follicles with giant cells in the adjacent lymphoid tissue.

A lymph-gland from the mesentery shows no abnormal features.

Pituitary Gland.—Owing to the difficulty experienced in removing this gland without damaging the surrounding bones, the section of the pituitary is incomplete.

Pars anterior: In a typical area there are few chromophil cells, but the basophil cells are most numerous. Clusters of acidophil cells occur but are not numerous. There are, however, areas in which acidophil cells predominate. *Pars intermedia*: Between the pars anterior and the pars nervosa there is a series of large vesicles containing friable homogeneous material resembling colloid. One large elongated vesicle, after fixation and sectioning, is 3·1 mm. long. Two other large vesicles encroach on to the pars anterior, whilst one 1·2 mm. in diameter is embedded in the pars nervosa. The *pars nervosa*, which is not complete, shows no abnormality apart from the large colloid vesicle mentioned above.

THE MACERATED SKULL

(*Museum Royal College of Surgeons of England.*)

The skull as a whole is greatly enlarged and very heavy. Without the mandible it weighs 3054 g. The mandible weighs 315 g.

Norma Frontalis (*Fig.* 101).—With the mandible in position the anterior aspect of the skull has an unusually rectangular shape owing to the large size of the mandible and of the zygomatic bones. The great width of the posterior portion of the skull can be appreciated on viewing the skull from the front. The protuberance of this portion is greater on the left side.

The frontal bone is 2·25 cm. thick in its thinnest part and has a finely pitted and grooved surface. No frontal suture is present. The supra-orbital margins are sharp and distinct. The glabella projects very little beyond the level of the nasofrontal suture. The nasofrontal and frontomaxillary sutures are plainly seen. The supra-orbital notch is shallow on the left side, but on the right side it is an almost complete foramen. The left supra-orbital notch is nearer the midline.

The Orbits.—The orbital apertures are irregularly oval in outline. The supra-orbital borders are sharp, but the lateral portions of the inferior margins are rounded owing to the thickening of the zygomatic bones. The fossæ of the lacrimal sacs are well defined. The posterior lacrimal crest on the right side is unusually prominent and on this side there is a small tuberosity on the maxilla, 6·25 mm. in diameter, at the inferior and lateral border of the fossa. The bone forming the roof of the orbit offers unusual resistance on palpation.

The medial walls are less parallel to each other than is usual, so that they face not only laterally but also slightly forwards. The laminæ papyraceæ of the ethmoid bone are hard and resistant to palpation and are obviously much thickened, as are the lacrimal bones. The lateral walls of the orbits face forwards to a greater extent than usual. These changes in the medial and lateral walls have caused constriction of the deeper parts of the orbital fossæ and great narrowing of the superior and inferior orbital fissures. The optic foramina are also much reduced in diameter.

All the sutures in the orbital fossæ are distinct, with the exception of those between the orbital plates of the frontal bones and the great wing of the sphenoid: the latter are completely obliterated.

The nasal bones are thickened, widened, and lie nearly in the vertical (coronal) plane, their lower borders projecting forwards very little more than the upper borders. The upper ends of the two bones taken together measure 2·25 cm. from side to side. In its upper part, the suture between them is deviated towards the right side. The nasomaxillary sutures run vertically, and near their lower ends are 2·2 cm. apart.

The zygomatic bones are thickened so that their lateral surfaces are unusually prominent and convex in all directions. As mentioned above, the orbital borders are rounded. The thickness of these bones renders the

zygomatic processes of the temporal bones invisible when the skull is viewed from the front. All the sutures surrounding these bones are distinct.

The maxillæ show considerable deformity. The frontal processes are widened from side to side. The prominent, so that the suture lies in the depth of a groove. The bone on each side of the pyriform aperture is rounded so that the outline of the aperture is indistinct. No anterior nasal spine is present. The zygomatic and alveolar processes are greatly thickened. The surface

FIG. 101.—*Norma frontalis*, with mandible in position. Note the unusually rectangular shape of skull. Skull of child of similar age is apposed for comparison.

FIG. 102.—Grossly thickened and deformed mandible, the whole bone except the cranial portions of the condylar and coronoid processes being affected. The mandible of a child of similar age is placed beside the deformed bone for comparison.

anterior surfaces of these bones contain smooth protuberances, that on the left side being the more prominent. The prominence of this part is greater than that of the nasal bones. The infra-orbital foramina are plainly seen. The bones on each side of the intermaxillary suture are of the bones is smooth except for the presence of small vascular grooves and of minute pits which vary in depth. These latter are most numerous on the inferior surfaces of the zygomatic processes and the lateral surfaces of the alveolar processes.

Nasal Aperture.—The rounding of the borders of this aperture has already been referred to. Its outline retains its pyriform shape. The nasal cavity is constricted by the thickening of the bones which bound it. The palatine processes of the maxillæ are rotated so as to face forwards as well as upwards. The anterior ends of the inferior conchæ are fused with the maxillæ and are thick, hard, and resistant.

The mandible (*Fig.* 102) is grossly thickened and deformed. The thickening affects all portions except the cranial portions of the condyloid and coronoid processes. The alveolar margin has been almost completely obliterated. The great widening of the body of the mandible has occurred mainly external to the line of the teeth. The surface of the bone is finely pitted. The mandible weighs 315 g. and in the midline is 3 cm. thick in an anteroposterior direction. The right side of the body measures 4·4 cm. from side to side in the widest part, which is behind the second right molar tooth. The body of the mandible measures 4·85 cm. in a vertical direction in the plane of the first right molar tooth. In the midline it measures 4·0 cm. in this direction. The majority of the teeth are carious.

posterior portion of this process and the mastoid foramen is present.

The great wings of the sphenoid bone are much reduced in anteroposterior diameter, since, in their widest parts, they measure 6·5 mm. in this direction on the right side and 13·5 mm. on the left.

All parts of the zygomatic bones are thickened. The frontosphenoidal process is enlarged and measures 2·2 cm. in an anteroposterior direction in its narrowest part. The zygomaticofacial foramina are plainly seen. All sutures surrounding these bones are distinct.

Norma Verticalis.—The left side of the skull is longer than the right and the parietal tuberosity on the left side is about 2·5 cm. farther back than on the opposite side. The right side is more prominent than the left. The zygomatic processes cannot be seen from above. The parietal suture is not visible. A large parietal foramen is present on the right side but there is none on the left. The pitted appearance of the surface of the bones has been noted.

Norma Occipitalis.—When viewed from the back the skull is seen to be asymmetrical, since the mastoid process projects farther downwards on the left side than on the

FIG. 103.—*Norma lateralis.* Coronal and lambdoid sutures are almost completely obliterated. Enlarged malar bones: orbits face much more laterally than usual. Skull of child of similar age is apposed for comparison.

Norma Lateralis (*Fig.* 103).—When the skull is viewed from the side the flattening of the bones of the face can be appreciated.

The orbits are seen to face laterally to a greater degree than usual. The anterior portions of the alveolar processes of the maxillæ reach a lower level than the posterior portions.

The coronal and lambdoid sutures are almost completely obliterated, a faint indication of their lines being visible only in the inferior portions. The sutures limiting the squamous portion of the temporal bones are indistinct posteriorly, but in their upper and anterior portions they can be plainly seen. The suture between the squamous portion of the temporal bone and the great wing of the sphenoid lies in the depth of a furrow. The parietomastoid suture is obliterated. The zygomatic process of the temporal bone is only very slightly thickened—measuring in the narrowest part, 6·5 mm. in width in a vertical direction and 4·3 mm. from side to side. All other portions of this bone are very greatly thickened. The mastoid process is more blunt than usual, the posterior and inferior aspects having a fairly uniform convexity. There are deep vascular channels in the right. The squamous portion of the occipital bone shows this asymmetry. The oblique direction taken by the saw-cut when removing the upper part of the calvarium exaggerates the asymmetrical appearance. The all-but-complete obliteration of the lambdoidal suture has been noted. The surfaces of the occipital and parietal bones are finely channelled and pitted.

Norma Basalis (*Fig.* 104).—The hard palate is greatly thickened and its surface grooved and finely pitted. The sutures in it are well defined. The foramen incisivum and greater palatine foramina are plainly seen. The posterior edge of the hard palate is rounded and the posterior nasal spine is thick and blunt. The alveolar processes of the maxillæ are thickened. The majority of the teeth are carious. The zygomatic processes of the maxillæ are seen to be enormously thickened. The inferior orbital fissures have become very much narrowed by the thickening of the bone on either side of them. Both pterygoid laminæ are grossly thickened. The choanæ (posterior nares) are much reduced in size by the thickening of the nasal septum, of the palatine bones, and of the pterygoid processes of the sphenoid bone. The right choana measures 1·75 cm. in a vertical direction and

0·65 cm. from side to side in its widest part. On the left side the measurements are 1·2 cm. and 0·65 cm. Inside the nasal cavity thickening of the inferior conchæ greatly reduces the size of these spaces.

The zygomatic processes of the temporal bones are not obviously thickened. Both glenoid fossæ are deeper than usual.

The openings of the left foramen ovale and left foramen spinosum are very much narrowed, but on the right side the openings are completely hidden from view by the thickening of the sphenoid bone.

The inferior surfaces of the petrous portions of the temporal bones contain numerous blunt projections, one of which, on either side, apparently represents a much thickened and abbreviated styloid process. The carotid canals are reduced in diameter and are partly hidden by thickening of adjacent parts of the occipital bone. The jugular fossæ are greatly reduced in size. The mastoid processes are thick and relatively blunt and the mastoid notches are shallow. The left mastoid foramen is large and deep and vascular channels are found in the surface of the adjacent bone. The right foramen is more nearly of normal size. The occipitomastoid sutures are obliterated.

The thickening of the occipital bone causes this to project downwards far beyond the level of the occipital condyles. The condylar canals are obliterated. The inferior nuchal lines are ill-defined. The external occipital protuberance and the superior nuchal line are inconspicuous.

The foramen magnum is reduced in size and measures 2·3 cm. in an anteroposterior direction and 1·9 cm. from side to side.

Section of the Cranial Vault.—The bone, on section, is seen to be enormously thickened. The thickest part which is found in the frontal bone where the crest for the attachment of the falx cerebri adds to the thickness, measures 3·92 cm. In other parts the bone varies from 1·37 cm. to 2·27 cm. in thickness. No diploë is seen. The cut surface has an ivory-like appearance and is marked by innumerable Haversian canals, which appears as minute brown flecks. These vary in size and number in different parts of the section.

Basis Cranii Interna.—The lack of symmetery seen in the outside of the skull is even more pronounced when the interior of the skull is inspected. The posterior boundaries of the three cranial fossæ are placed farther back on the left side, as are most of the landmarks they contain.

The anterior fossa extends farther forward on the right side and farther back on the left.

The crista galli of the ethmoid bone is thickened and lobluated. The adjacent portions of the orbital plates of the frontal bone come into contact with the crista galli and so almost completely conceal the lamina cribrosa of the ethmoid. This latter structure, though still perforated by minute apertures, is thick and resistant to palpation. The frontal crest is thicker and more prominent than usual and the lower extremity of it is divided into two small lobes. The orbital plate of the frontal bone is very dense and much thickened and the surface contains many blunt bosses and ridges. No suture can be seen between this bone and the small wings of the sphenoid. These wings are obviously thickened and have rounded edges and blunt anterior clinoid processes.

Middle Fossa.—This appears to be reduced in size by the numerous lumps and ridges present in the bones forming the floor. These are particularly prominent on the right side.

The tuberculum sellæ is unusually prominent and well defined. The optic foramina are slightly reduced in diameter. The fossa hypophyseos is not obviously reduced in size and the dorsum sellæ and posterior clinoid processes are not thickened. The foramina lacera are partly obliterated by union between the summits of the petrous portions of the temporal bones and the opposing parts of the great wings of the sphenoid. The

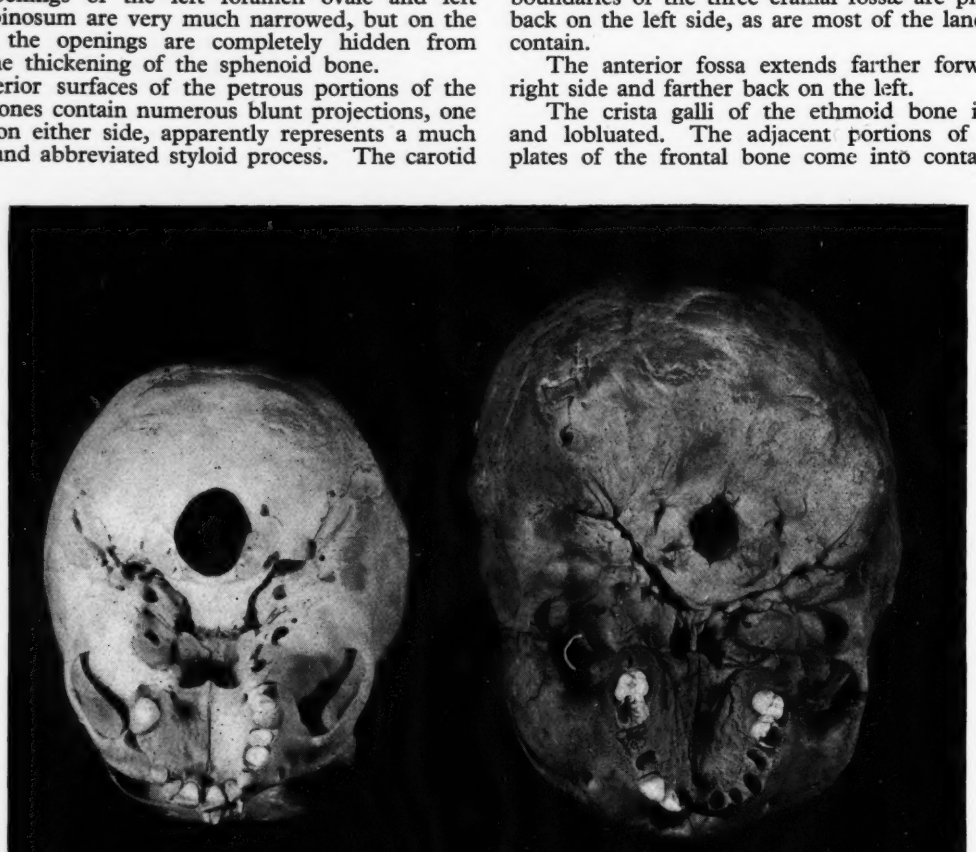

FIG. 104.—*Norma basalis.* A skull of a child of the same age is placed alongside for purposes of comparison.

widest parts of the foramina, which transmit the carotid arteries, are patent and well-formed canals persist in the bone on either side of the body of the sphenoid, where their circular apertures may be seen. The lateral sides of these canals are formed of sharp ridges, slight processes in which indicate the lines of fusion of the two bones. The lateral extremities of the foramina lacera are indicated by narrow fissures immediately posterior to the foramina ovalia. The foramen ovale on each side is reduced in diameter, especially in the deeper portions. The right rotundum is much reduced in size, but that on the left side is of about normal size. The superior orbital fissures are very much narrowed by the thickening of the greater and lesser wings of the sphenoid bone.

The internal orifice of the left foramen spinosum is hidden from view by the overhanging edges of the bone in front of and behind the orifice. A thread passed through the foramen from the base of the skull emerges on the lateral side of and slightly behind the foramen ovale in the depth of a deep depression.

The groove for the middle meningeal artery is, for about 5 mm., extremely deep but not completely bridged over by bone.

Lateral to this, for the distance of about 2·5 cm., the groove is converted into a canal formed by the apposition of the bone on either side, the line of apposition being indicated by a furrow. Lateral to this canal the groove again becomes open for a distance of 2 cm., but again sinks into the bone. Where the saw-cut crossed the canal it is almost in the centre of the very much thickened bone. Branches of the artery also run in deep grooves or closed canals. The course of the right middle meningeal artery is also partly obscured by projection and apposition of bone, which has converted the groove for this artery into a canal 2·3 cm. in length. In the upper portions the groove is deep but is not completely surrounded by bone.

The anterior surfaces of the petrous portions of the temporal bones are unusually lumpy. The depression for the semilunar ganglion is deepest on the right side, and on this side the bony eminences are more prominent. A deep groove for the superior petrosal sinus is present on this side, but no such marking is seen on the left side.

The posterior fossa is diminished in size by the presence of bony protuberances on the occipital bone and on the posterior surfaces of the petrous portions of the temporal bones. The foramen magnum is diminished in size, as noted above. When the skull is seen from above the openings of the hypoglossal canals are hidden from view by projections of the bone above them, but when the skull is tilted the openings are seen to be reduced in diameter.

The internal acoustic meatus on each side is funnel-shaped and constricted. Bony swellings completely hide both jugular foramina. The groove for the left transverse sinus is larger and deeper than that for the right; those for both sigmoid sinuses are also deep, but on the left side the groove is converted into a long canal. Leading from the medial portions of these grooves are two smaller grooves which run into the foramen magnum. On the right side a smaller groove runs from the medial part of the groove for the sigmoid sinus into the opening of the hypoglossal canal. Grooves for the superior petrosal sinuses are shallow, but furrows in the bone in this region indicate the site of the suture between the petrous parts of the temporal bones and the basal part of the occipital bone.

The internal occipital protuberance is unusually prominent, as is the ridge for the attachment of the falx cerebelli. A deep groove runs along this ridge from the internal occipital protuberance to the foramen magnum.

The interior of the cranial vault is unusually lumpy and contains deep vascular channels. A deep furrow containing many pits marks the line of the coronal suture, which is now obliterated. The frontal crest is thick and prominent and continues upwards to a point about half-way between its commencement and the line of the coronal suture. A groove for the superior longitudinal sinus is plainly seen, especially in its posterior part.

Measurements.—

Maximum length (glabella to maximal occipital point), 20·3 cm.
Maximum width (between parietal tuberosities), 16·3 cm.
Bi-asterionic width, 14·2 cm.
Bi-stephanionic width, 13·5 cm.
Bi-zygomatic width, 12·7 cm.
Height of cranion (basion to bregma), 15·9 cm.
Horizontal circumference, 62·5 cm.
Longitudinal arc, 00·0 —.
Nasion to progonion (mental tubercle), 12·3 cm.
Nasion to prosthion (alveolar point), 7·5 cm.
Basi-alveolar length, 8·5 cm.
Basi-nasal length, 8·4 cm.
Nasal width, 2·3 cm. (rounded edges).
Nasal height, 5·1 cm. (rounded lower edge).
Orbital height (left side), 3·4 cm.
Orbital height (right side), 3·1 cm.
Orbital width (left side), 3·6 cm.
Orbital width (right side), 3·7 cm.
Palatomaxillary width, 6·1 cm.
Palatomaxillary length, 4·9 cm.

$$\text{Vertical index} = \frac{\text{Height} \times 100}{\text{Length}} = \frac{15{\cdot}9 \times 100}{20{\cdot}3} = 78{\cdot}3$$

$$\text{Total facial index} = \frac{\text{Nasomental length} \times 100}{\text{Bi-zygomatic width}} = \frac{12{\cdot}3 \times 100}{12{\cdot}7} = 96{\cdot}8$$

$$\text{Superior facial index} = \frac{\text{Naso-alveolar length} \times 100}{\text{Bi-zygomatic width}} = \frac{7{\cdot}5 \times 100}{12{\cdot}7} = 59$$

$$\text{Gnathic index} = \frac{\text{Basi-alveolar length} \times 100}{\text{Basi-nasal length}} = \frac{8{\cdot}5 \times 100}{8{\cdot}4} = 101$$

$$\text{Nasal index} = \frac{\text{Nasal width} \times 100}{\text{Nasal height}} = \frac{2{\cdot}3 \times 100}{5{\cdot}1} = 45$$

$$\text{Orbital index (left)} = \frac{\text{Orbital height} \times 100}{\text{Orbital width}} = \frac{3{\cdot}4 \times 100}{3{\cdot}6} = 94{\cdot}4$$

$$\text{Orbital index (right)} = \frac{\text{Orbital height} \times 100}{\text{Orbital width}} = \frac{3{\cdot}1 \times 100}{3{\cdot}7} = 83{\cdot}7$$

$$\text{Palatomaxillary index} = \frac{\text{Palatomaxillary width} \times 100}{\text{Palatomaxillary length}} = \frac{6{\cdot}1 \times 100}{4{\cdot}9} = 124{\cdot}4$$

OTHER BONES

Right Clavicle.—The right clavicle is much thicker and heavier than a normal bone, the thickening being most pronounced in the medial and lateral thirds. The sternal end is very little larger than the adjacent part of the shaft of the bone. The shaft exhibits the usual double curve, but the various borders and tuberosities have become ill-defined. Small areas of roughening represent the deltoid tubercle and the tuberositas coracoidea. The greater part of the surface is smooth and ivory-like, but at both ends there are small zones which are slightly roughened and more nearly resemble the surface of a normal bone. The clavicle weighs 51 g. when macerated.

Left Clavicle.—This was bisected by a horizontal longitudinal section. The greater part of the bone is very dense and no central spongy bone is present, but the Haversian canals, which can be seen as small longitudinal streaks, are slightly larger near the centre of the

bone and also in a blood-stained area in the anterior portion of the medial half. There are areas at each end where the bone is much less dense, though not quite as spongy as in a normal bone. This area is triangular at the sternal end, the base of the triangle abutting on the articular cartilage. A similar area is found at the acromial end, and it was from this end that a microscopical section was taken so as to include both dense and relatively spongy bone.

Fourth Right Rib.—The posterior aspect of the head, neck, and tubercle show very little change, but the bone on the anterior and superior aspect of these structures is thick and smooth. Immediately lateral to the tubercle the bone becomes thick and rounded, the thickness being greatest in the region of the angle of the rib. The greater part of the shaft of the rib is thick and rounded, the bone having a very smooth hard surface. The thickness gradually diminishes as the shaft is traced towards the point of the attachment of the costal cartilage, where the bone is of almost normal shape and size. Here the surface has the faint roughness and the minute longitudinal furrows seen in a normal rib. The rib weighs 34 g. when macerated.

Fifth Right Rib.—The greater part of the 5th right rib has been cut in a longitudinal direction. It is greatly thickened, being 1·5 cm. thick in the widest part, which is at the angle. The bone is very dense, but in the central portion of the shaft the Haversian canals are larger and more closely set. There is a zone of spongy bone in the posterior portions of the head, neck, and tubercle abutting on the articular cartilage of the tubercle. The articular cartilage of the head of the rib was not preserved. Spongy bone is also found in the centre of the anterior portion of the shaft of the rib.

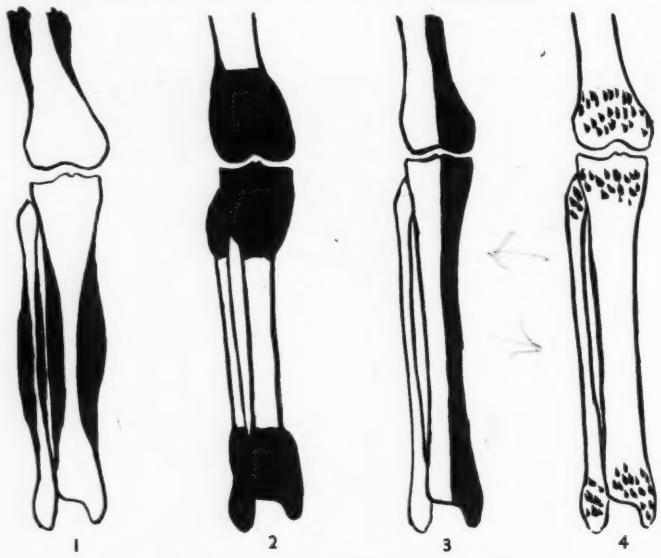

FIG. 105.—Diagrammatically depicts the four dystrophies discussed: (1) Engelmann's disease; (2) Osteopetrosis (or 'marble bones'); (3) Osteopœcilia (or 'spotted bones'); (4) Melorheostosis (Leri's disease or Junghagen's monomeric eburneous osteosis).

HISTOLOGY OF THE BONES

Skull.—The section of the skull consists entirely of compact lamellated bone with Haversian canals of varying sizes. The soft tissue in the canals is not well preserved, but in many it appears to be unusually fibrous, though not large in amount. In some canals the bone is bounded by osteoblasts. No diploë is present.

Left Clavicle.—The section present was taken from the acromial end through the junction of the very dense and compact bone with some spongy bone which is present near the articular cartilage. The compact part resembles that seen in the skull, but the osteoblasts lining the Haversian canals are more numerous and closely set in the present section. Two small Haversian canals each contain two large osteoblasts and some larger canals contain similar but smaller cells. The periosteum of this portion has become partly separated during preparation of the section. The outer part is dense, fibrous, and lowly cellular. Internal to this is a more cellular layer of fibrous tissue, the fibres of which run parallel to the bone. These two layers have become separated from an inner layer which is still attached to the bone. The latter is formed of ovoid or cuboidal osteoblasts, which form an almost continuous layer that in parts is several cells thick.

The transition between the compact and spongy portions is abrupt. Bounding the compact portion is a continuous layer of osteoblasts which are closely packed, many being columnar in shape.

The spongy portion is composed of bony trabeculæ supporting fatty and cellular marrow. Some of these are partly surrounded by prominent osteoblasts, but others by osteoblasts which are flat and inconspicuous.

In all parts of the section the osteoblasts are found only in contact with bone—either lining the Haversian canals or surrounding lamellæ. No osteoid tissue is recognized since after decalcification the whole of the bone has a uniform pink colour with hæmatoxylin and eosin staining.

Vertebral Body.—This section shows little abnormality. There is no over-activity of osteoblasts and those attached to the trabeculæ are not unusually large or numerous.

DISCUSSION

Congenital bone dystrophies associated with unusual osteosclerosis may be grouped into four main groups:—

1. Engelmann's disease (or osteopathia hyperostotica sclerotisans multiplex infantilis).
2. Osteopetrosis (or marble bones.)
3. Osteopœcilia (or spotted bones).
4. Melorheostosis (Leri's disease or Junghagen's monomeric eburneous osteosis). Spotted epiphysis has been purposely omitted from this group.

The main features of these four dystrophies are depicted in the accompanying diagram (*Fig.* 105).

The case under consideration does not present the classical features of any of these conditions. Cases somewhat similar to this child have been reported by S. Hirsch and by Ivimay. In the latter's case only one side of the skull was involved, and the diagnosis reached was that the condition was a combination of leontiasis ossea, osteitis deformans, and osteitis fibrosa.

Certain features of the child described in this communication were briefly discussed in an article on leontiasis ossea (Australian Medical Congress, January, 1934). It was pointed out that the skull presented features indistinguishable from those of the diffuse osteitic type of leontiasis ossea; and the suggestion was made at the time that it would be better to classify this case, together with the cases of S. Hirsch and Ivimay, as a separate bony dystrophy.

The radiographs of this child's skull would appear to be identical with Jadelot's skull, a skull dug up in France, the other bones of the skeleton not being recovered. Knaggs classifies this skull as being of the generalized osteitic type of leontiasis ossea.

In brief, this patient's skeleton presents features of bony dystrophy, apparently congenital, which does not conform to osteopetrosis, but which certainly cannot be regarded as Engelmann's disease; and it would seem better to assign it, with the cases of S. Hirsch and Ivimay, to a separate group under an individual name.

The author is greatly indebted to Dr. Sear and Dr. Hansman for their radiological and biochemical reports, and particularly are his thanks due to Dr. Davies, whose thorough investigation of the autopsy findings has given this communication its chief value.

MASKED STEATORRHŒA REVEALED BY PSEUDO-FRACTURES (LOOSER'S ZONES)

WITH SOME OBSERVATIONS ON CALCIUM EXCRETION IN CONVALESCENCE

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In 1920 Looser described the clinical picture and the radiological and histopathological findings in 23 cases of osteomalacia from various causes. He made special reference to the "*merkwürdigen Pseudofrakturen*" (remarkable pseudofractures) and "*Umbauzonen*" (zones of transformation) in apparently normal bone. These appearances he described in some detail, recognizing clearly that they were not peculiar to any one disease but could occur in any condition involving continuous failure of adequate deposition of calcium in the skeleton. Milkman (1930, 1934) described as a new disease entity the condition of a woman followed over eight years who showed similar lesions in her skeleton. This patient gave a history of renal calculi in 1920 and 1922, and in 1931 she was found to have intermittent glycosuria and ketonuria. On the grounds that there was no evidence of glandular or dietetic deficiency, and, further, because the lesions failed to heal on a diet supplemented by vitamin therapy, Milkman rejected the diagnosis of osteomalacia. He does not appear to have considered the possibility of renal changes leading to osteomalacia although at autopsy it was established that the woman had suffered from a diffuse nephritis with arteriosclerosis. Bennett, Hunter, and Vaughan (1932) in their study of 15 cases of steatorrhœa described the associated radiological appearances in some detail, but though osteomalacia occurred they made no mention of Looser's lesions.

The first case reported in the British literature to show skeletal lesions analogous to those reported by Looser and by Milkman was that of a girl of 24 described by Leedham-Green and Golding (1937). They suggested the term 'osteoporosis melolytica' as "indicating an association of a definite type of pathological change in the skeletal system with an elective affinity of the process for the bones of the limbs". Edeiken and Schneeberg (1943) describe a woman of 34 having similar radiological findings who had had evidence of bone disease from infancy and was, when they observed her, in negative calcium balance. MacKay and May (1945) reported two children suffering from rickets with multiple fractures in whom healing did not occur until the oral dosage of vitamin D had been raised to over 400,000 units daily. In 1946 Albright, Burnett, Parson, Reifenstein, and Roos in a comprehensive article discussing osteomalacia and late rickets, described 9 cases of osteomalacia, 2 of which had steatorrhœa, showing bone lesions similar to those outlined by Milkman. McCance (1947) reported a further case of a girl aged 17 with multiple symmetrical fractures in whom the calcium balance became positive only when the oral dosage of vitamin D had been raised to 500,000 units daily. Though only one fæcal analysis is reported, a personal communication makes it clear that the writer was at pains to exclude steatorrhœa. The patient described by Short (1948) was a middle-aged woman in whom there were gross skeletal changes and multiple pseudo-fractures associated with faulty fat absorption.

The following case is described to show how pseudo-fractures and the disability arising therefrom, in the absence of any gross deformity, may dominate the clinical picture. There was no history of bowel dysfunction, nor were the stools abnormal on visual examination.

CASE REPORT

History.—An unmarried female, aged 22 years, first attended the Royal National Orthopædic Hospital in March, 1945, complaining of painful feet, which had become gradually worse for the past year. A diagnosis of metatarsalgia was made and she was given physical

treatment, but without benefit. She did not attend the hospital again until January, 1948, when she stated that her symptoms had remained unchanged and that in 1947 she had been seen at another hospital for a fracture of her left foot. She was now referred to St. George's Hospital for more intensive study and when seen there in February, 1948, the pain in her feet had been continuous for four years. It had started in the left foot and, although it was now present in both, the left was still the more affected. The pain was worse on walking, more intense in the winter, and accompanied by numbness and a sensation of pins and needles. She had difficulty

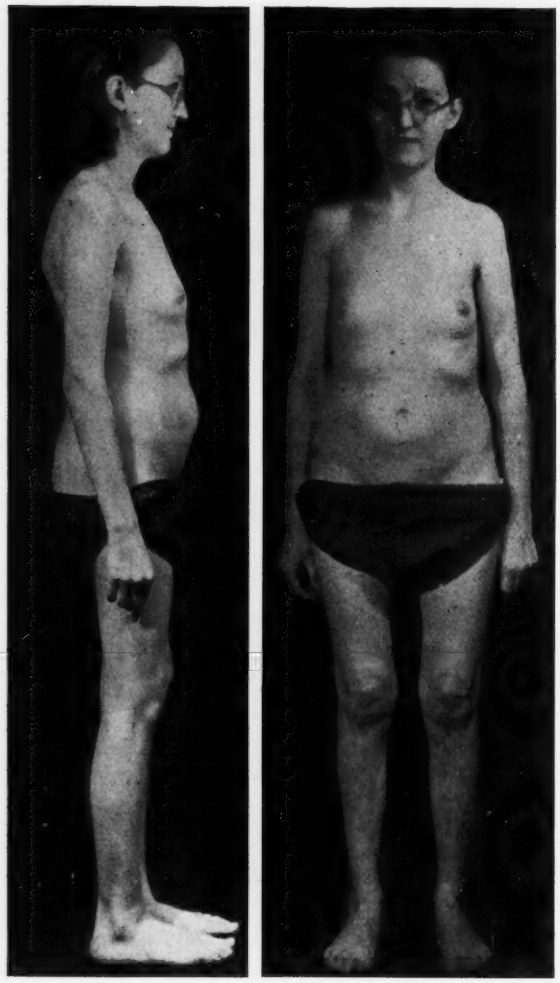


FIG. 106.—Photographs of patient.

in walking and rising from the supine to the erect position, but she stated that her general health was otherwise good, her appetite fair, and her bowels, micturition, and menstruation entirely normal. She had not been losing weight.

She gave no significant information as to her past medical history, stating only that she had had measles, chicken-pox, tonsillectomy, and a fracture of one of her right toes. Her mother, however, subsequently informed us that when the patient was 18 months old she was told at an infant welfare centre that the baby had rickets, and a course of ultra-violet light and extra cod-liver oil were prescribed. The mother also stated that the child had never in her life been constipated and had often had two and sometimes three apparently normal stools a day. She added that she had brought up a large family and that the children were all big and healthy. The family economy and diet had always been adequate and therefore the mention of rickets in this case had surprised her. The family history had no significant features, both parents and seven siblings being in good health.

ON EXAMINATION (*Fig.* 106).—The patient was found to be an intelligent young woman with pallid features. Her breasts were under-developed and her build was that of a girl of 15 years. There was slight protuberance of the abdomen. The mouth and throat were normal and the teeth in good condition. The cardiovascular and respiratory systems showed no abnormality and her blood-pressure was 110/70 mm. The liver and spleen were not palpable. Reflexes were normal and Trousseau's and Chvostek's signs negative. There was tenderness to pressure over the ribs, pelvis, and feet. Temperature and pulsation in the extremities were normal. Her weight was 6 st. 6 lb. She was noted to have a waddling gait and when asked to get up from the supine position unaided she had considerable difficulty, her movements being reminiscent of a case of muscular dystrophy.

Radiological examination of the skeleton showed generalized osteoporosis. Looser's zones were noted in the shafts of the 3rd and 4th metatarsals of the left foot (*Fig.* 107A) and in the shafts of the 4th and 5th metatarsals of the right foot. The pelvis showed an early stage of triradiate deformity, and Looser's zones were seen in the superior and inferior rami of the os pubis on the left side and the inferior ramus on the right side (*Fig.* 108). There were small translucent areas on the axillary borders of both scapulæ (*Fig.* 109A).

Examination of the *peripheral blood* gave the following results :—

Red blood-corpuscles	3,760,000 per c.mm.
White blood-corpuscles	6500 per c.mm.
Hæmoglobin	83 per cent (Haldane)
Colour index	1·11
Mean corpuscular volume	112 cu.μ
Mean corpuscular diameter	8·1μ
Packed-cell volume	42 per cent
Neutrophils	2952 per c.mm.
Lymphocytes	2952 per c.mm.
Monocytes	390 per c.mm.
Eosinophils	260 per c.mm.
Prothrombin time	10·5 sec.
E.S.R. (Wintrobe)	22 mm. in 1 hr.

The Wassermann reaction was negative.

Bone-marrow material obtained by sternal puncture showed well-marked normoblastic hyperplasia. Megaloblasts of Ehrlich were not seen. Megakaryocytes were numerous and the white cells and their precursors were normal.

Repeated examinations of the *urine* were negative for proteins and reducing substances and chemical examinations of the *blood* and *fæces* gave the following results :—

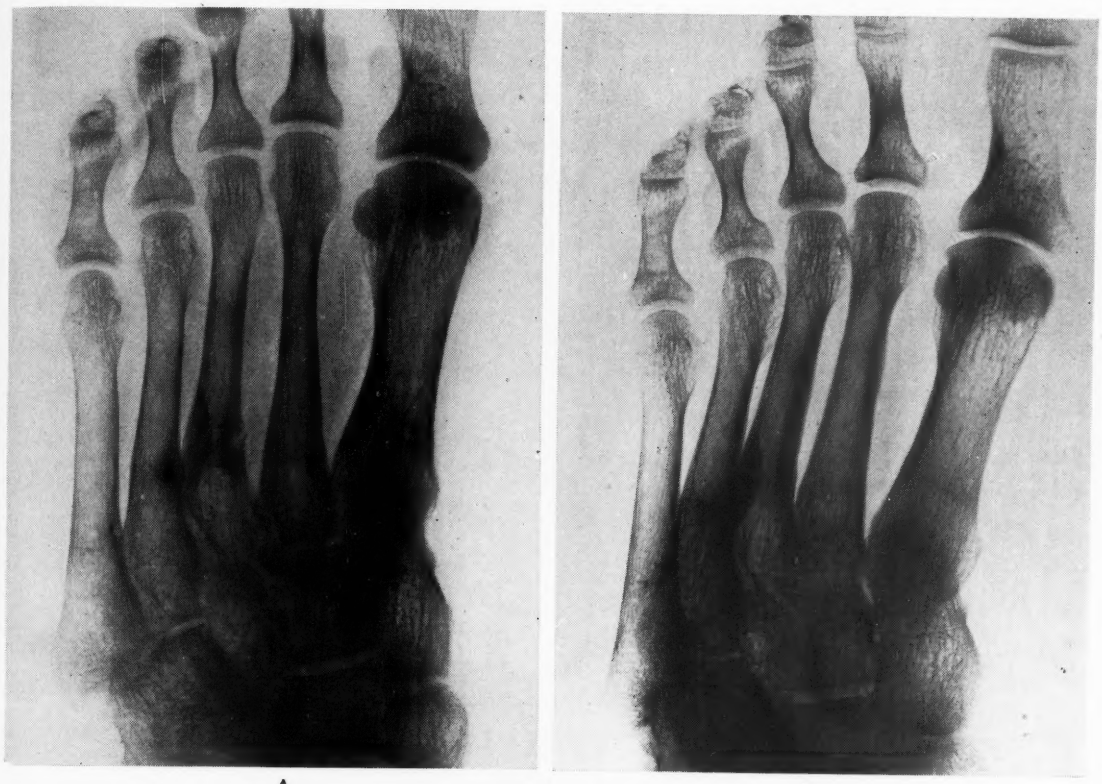
Serum-calcium	4·85 mEq. (9·7 mg. per cent)
Serum-inorganic-phosphorus	1·2 mEq. (2·1 mg. per cent)
Blood-urea	25 mg. per 100 ml.
Alkaline phosphatase	55 units
Total fæcal fat 35 per cent	Split fat 76 per cent Unsplit fat 24 per cent
Fractional test-meal	(*See Fig.* 110)

These and subsequent biochemical findings are grouped in the tables for clarity.

The results of investigations show the typical flat blood-sugar curve of idiopathic steatorrhœa. The intravenous curve is normal (*Table III*). The fault in fat absorption is clearly shown in *Table II*. Thus on an intake of 75 g. before treatment was commenced there was an absorption of 84·4 and 77·7 per cent, and on a restricted intake of 40 g. during treatment and of 50 g. during the convalescent period the fault was even more clearly marked, only 58·0 and 56·5 per cent being absorbed.

COURSE AND RESPONSE TO TREATMENT.—The patient was placed on a normal diet with fats restricted to 40 g. per day. She was given 150,000 units of vitamin D in

the form of calciferol and calcium in the form of tribasic calcium phosphate, 1 g. four times a day. The drugs were administered between meals. She was also given a course of ultra-violet light. The response to treatment was dramatic; she was free from pain within a fortnight and had practically no disability in getting up and walking within six weeks. Her radiographs also showed evidence of rapid recalcification. The blood-picture was, however, unaltered and she was therefore given intramuscular injections of a crude liver preparation daily for the first week and then every third day, but still without improvement. She was discharged from hospital towards the end of May, symptom-free, and her vitamin-D intake was then gradually reduced to 15,000 units by mouth daily and the diet and calcium phosphate intake left as before. Associated with the recalcification there was a progressive drop in the level of alkaline phosphatase and a rise in the serum-inorganic-phosphorus; these changes are shown in *Table I*, where the principal serum analyses are grouped together.



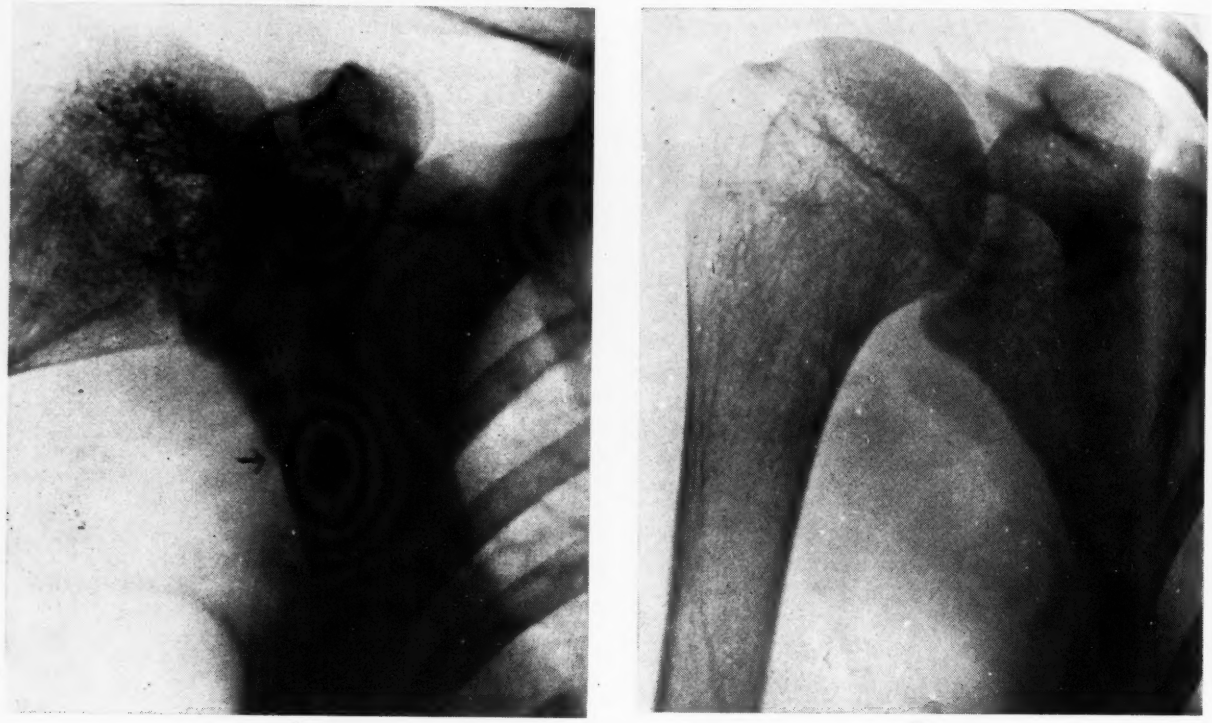
A B

FIG. 107.—A, Showing Looser's zones in first, third, and fourth metatarsals; B, Healing 12 weeks later.

FIG. 108.—Radiograph showing osteoporotic pelvis with Looser's zones.

While we were confident that we were maintaining an adequate intake of vitamin D during the eight weeks' treatment, we felt it advisable to ensure an adequate supply of other fat-soluble vitamins. Alpha-tocopherol

was therefore prescribed in doses of 150 mg. thrice daily. After four weeks on this treatment the patient remarked on the real improvement in her strength and



A B

FIG. 109.—A, Area of rarefaction in axillary border of right scapula; B, Healing 12 weeks later.

general well-being. This observation was, to us, a striking confirmation of Albright's findings (1946) on his patient No. 395554, Case No. 2, who showed similar marked improvement.

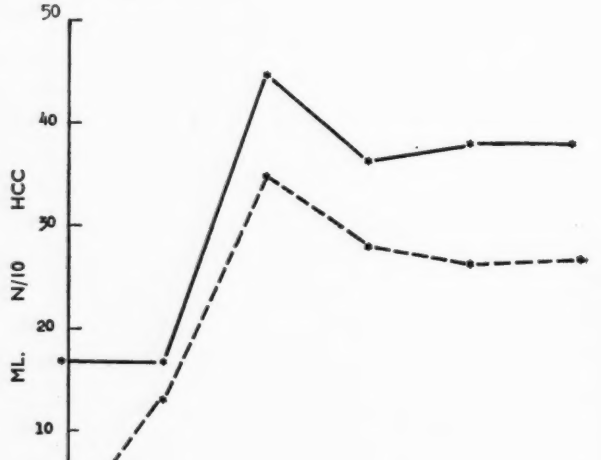


FIG. 110.—Fractional test-meal, March 8, 1948. Stimulus, 100 ml. 7 per cent alcohol in water.

During the convalescent period, when the full co-operation of the patient had been obtained, it was found possible to carry out a short balance study. For fourteen days before the four-day period of intensive study she was placed on a strictly controlled diet. The average fat excretion during the four-day period is given in *Table II*. The total calcium intake for the four-day period was 6000 mg. The calcium excreted in the fæces was 1580 mg. and in the urine 100 mg. Hence the total calcium retained was 4320 mg., giving a daily retention of calcium of 1080 mg., which is of the order of 25 mg. per kilo body-weight per day (patient's weight, 44 kg.). It would seem therefore, that recalcification was still progressing vigorously some three months after therapy had been started and even though there was a marked drop in the circulating phosphatase level.

DISCUSSION

This case demonstrates that a failure in fat absorption sufficient to produce incapacitating skeletal changes need be associated neither with any gross irregularity of bowel action nor with any abnormality of the stools recognizable on gross examination. Neither Leedham-Green and Golding (1937) nor Edeiken and Schneeberg (1943) mention having carried out fat analysis of the stools—nor do MacKay and May (1945) give any figures of analysis to substantiate their claim that there was no fault in fat absorption in their two cases. The failure to achieve calcification with normal therapeutic doses of vitamin D by mouth does not exclude steatorrhœa, for it is in just such cases that an exceptionally high dosage may be needed to be effective. Fourman and Spray (1948), in their investigation of vitamin-D absorption in steatorrhœa, well illustrate this point and in addition demonstrate that " parenteral therapy with ordinary doses of vitamin D is effective whereas oral therapy requires very large dosage and is at best uncertain ". Continued parenteral therapy may be

a definite inconvenience to a patient, and we have noted that after an initial period of massive oral therapy we have been able to diminish the dosage of vitamin D to 15,000 units a day and still maintain a positive calcium balance without regression in the patient's condition. We suggest that a division be made between those cases of rickets which are clearly resistant to parenteral therapy (Albright, Butler, and Bloomberg, 1937) and those which are resistant to oral therapy only. It is clear from our case that the assumption that there is no fault in fat absorption to account for resistance to oral vitamin D requires a more detailed study than has been made in many of the reported cases. Once the intestinal barrier is breached and adequate calcium absorption assured, the mechanism of retention of the circulating calcium in the body arises. Bauer (1929) and his colleagues studied 13 normal men on a restricted calcium intake of 0·33 g. calcium over each three-day period. They found an average total urinary output of 0·19 g. for each three-day period. Rose, quoted by the above authors, studying persons on a diet containing 1·021 g. per three-day period, found a urinary output of 0·218 g. for three days. Aub and his collaborators (1932) studied a woman suffering from steatorrhœa, and in her found an output of 0·010 g. over a three-day period. They attributed this low figure to her serum-calcium level, which, being between 5 mg. and 6 mg. per 100 ml., was near or below the ' renal threshold ' for calcium. In our patient during convalescence the urinary calcium excretion over four days was equivalent to 0·075 g. per three-day period, with the *p*H of the urine varying between 6·7 and 7·1. Her serum-calcium during this period was at a level of 9·4 mg. to 9·6 mg. per 100 ml. It would appear, then, that one must postulate that the ' renal threshold ' to calcium is labile and that over the period of observation her kidneys were operating at a higher threshold level than usual. On examination, Bauer's series of normals seem to support this concept of the labile threshold : thus patient W. C., in period

Table I.—Results of Biochemical Analyses

	Date										
	March 2	March 4	March 9	March 10	March 15	April 22	April 29	May 8	May 30	June 28	July 27
Kations in serum (mEq.)											
Ca	4·85				4·8	4·8	5·0		4·8		4·7
Na					148·5						146·0
Anions in serum (mEq.)											
P	1·2		1·25		1·25		2·15	2·3		2·5	2·6
Cl			102·0		102·0		102·2				105·0
HCO_3			30·4		29·8		30·4				29·4
Phosphatase (King Armstrong units)											
Acid				4·6			4·4			3·3	3·9
Alkaline			55·5	54·5		36·0	31·2	33·0	33·0	24·0	19·6
Urea, fasting (mg./100 ml.)			25·0				27·0			34·0	30·0
Glucose (fasting)											
Cap. blood		95·0				93·0					81·0
Protein (mg./100 ml.)							7·4			7·7	7·5
Albumin							3·9				
Globulin							3·5				
Carotenoids (I.U.) (Normal range, 80–160)				70·0				39·0		50·0	65·0
Vitamin A (I.U.) (Normal range 80–160)				86·0				95·0		65·0	165·0

Table II.—Fat and Calcium Analyses

	Before Treatment		During Treatment Average of 3-day Period	Convalescent Average of 4-day Period
Date	March 9, 1948	March 23, 1948	April 23/25, 1948	July 27/31, 1948
A. Fat Analysis.—				
Intake g.	75	75	40	50
Output total in g.	11·7	18·2	16·9	21·75
Percentage absorbed	84·4	77·7	58·0	56·5
Percentage of fæces	44·0	53·0	37·5	41·0
Percentage split	76·0	77·0	80·5	72·0
Percentage unsplit	24·0	23·0	19·5	28·0
Absorbed g. per kilo body-weight	1·54	1·4	0·75	0·63
B. Calcium Analysis.—				
Intake by mouth	1·0	1·0	5·7	1·500
Output in fæces	0·69	0·59	3·7	0·395
Absorbed	0·31	0·41	2·0	1·105
' Absorbed ' g. per kilo body-weight	0·007	0·008	0·049	0·029

3 excreted only 0·040 g. of calcium in three days, and patient D. M. in period 17 excreted 0·05 g. of calcium. Both were on restricted intake, but presumably their blood-serum-calcium levels were normal or well above the normal 'threshold'.

Circumstances prevented us from carrying out as adequate a study of our patient as we would have desired, but so far as they go the results demonstrate clearly faults in fat and in carbohydrate absorption (*Table III*), neither of which has been significantly improved, although the patient has shown marked clinical and radiological improvement. With evidence of recalcification the level of inorganic phosphorus in the blood has risen and the alkaline-phosphatase level has dropped, though it is still above normal. Associated with the high alkaline phosphatase there has been some elevation of the acid phosphatase; this has tended to fall with the alkaline phosphatase, but is still somewhat high. The anæmia likewise has been inconsistent in response to therapy, a finding which is confirmed by others and recently stressed by Cooke, Frazer, Peeney, Sammons, and Thompson (1948).

Table III.—GLUCOSE-TOLERANCE, VAN SLYKE, AND GASTRIC TESTS

Glucose-tolerance Curves.—Capillary blood-sugar levels. Time from administration of glucose in brackets.

	March 4	April 22	July 27
	Minutes	*Minutes*	*Minutes*
Oral.—			
50 g. glucose given	96 fasting	93 fasting	103 fasting
	76 (30)	114 (11)	99 (15)
	126 (90)	117 (41)	105 (30)
	105 (120)	119 (71)	119 (64)
	129 (150)	117 (101)	105 (89)
			81 (121)
			80 (148)
Intravenous.—			
Test dose 20 g.	106 fasting		
	185 (15)		
	138 (30)		
	131 (45)		
	112 (60)		
	114 (75)		

No reducing substances found in the urine during any of the tests.

Van Slyke Clearance Tests.—
100 per cent normal, C maximum, March 10.
98 per cent normal, C standard, July 27.

Gastric Test-meal.—*See Fig.* 110.

The radiological appearances and bone changes which Milkman described were recognized by German and Austrian workers as occurring in cases of hunger osteomalacia following the 1914–18 war and were described in detail by Looser (1920), who accounted for their symmetrical occurrence by the fact that stresses and strains are apt to be symmetrical in distribution, whereas trauma is not. Brailsford (1948), in his book, includes Milkman's case with those of osteomalacia. It was not apparently sufficiently appreciated by Milkman and others that osteomalacia could be the end-result of a varied aetiology, and it remained for Albright and his associates (1946), in their classical and most informative paper, to clarify the situation.

SUMMARY AND CONCLUSIONS

1. A case of osteomalacia due to steatorrhœa is described. It was revealed in the first instance only by the presence of Looser's zones in the bones of the feet. Pseudo-fractures were present in the feet, pelvis, and axillary border of the scapula. There was no history of abnormal bowel function or of any gross abnormality of the stools.

2. The case is discussed in relation to Milkman's syndrome and osteomalacia arising from resistance to vitamin D and renal acidosis.

3. It is pointed out that in any obscure osteomalacia which may only respond to high dosage of vitamin D, resistance to this vitamin cannot be assumed unless steatorrhœa has been excluded by thorough examination of the stools.

4. Calcium balance figures are presented and their significance is discussed.

Our thanks are due to Dr. Moore, of the Dunn National Laboratory, Cambridge, for carrying out the carotenoid and vitamin-A estimations.

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NON-SPECIFIC (EOSINOPHILIC) GRANULOMA OF BONE

By R. M. HILL, Carlisle

The condition described by American writers of recent years as 'eosinophilic granuloma of bone' has received conspicuously little mention in the British literature since the late Sir John Fraser's admirable review (1935), entitled 'Skeletal Lipoid Granulomatosis'. Following the publication by Otani and Ehrlich (1940) and an earlier work (1940) of their own, Jaffe and Lichtenstein (1944) awakened interest by a classical contribution in which they reviewed with effect many of the cases of this curious lesion published under such varying nomenclature as 'solitary granuloma of bone' (Otani and Ehrlich), 'non lipoid histiocytosis' (Letterer-Siwe disease), and 'lipogranulomatosis' (Hand-Schüller-Christian disease). These authors drew attention to the similarity in the pathological appearances at various stages of these conditions which they concluded to be expressions of the same fundamental disorder, and they were able in many instances to trace the transition from one to another group. At the same time they regarded the presence of eosinophils to be sufficiently characteristic of a stage in the process and in a particular type of granuloma as to constitute adequate grounds for the designation 'eosinophilic granuloma of bone'. Other views on this problem will be presented later. While it is not intended to subscribe a complete review of the literature, the comparative rarity of the condition, the scanty references in British works, and its importance as a feature in the differentiation from other lesions, in particular malignant tumours of bone, warrants a brief summary of our present knowledge in addition to the presentation of an individual case.

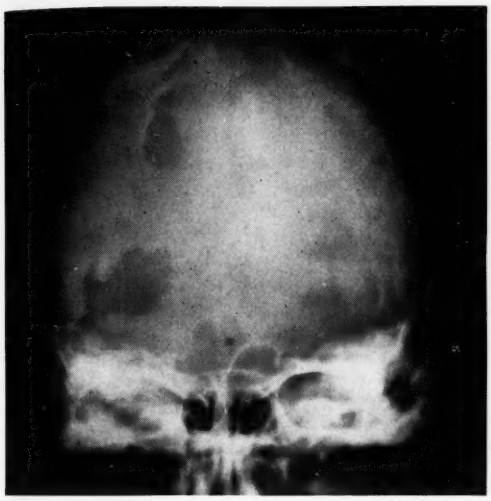

Fig. 111.—Initial appearance. Note the geographical outline of the area of rarefaction of the right frontal bone, the clear-cut edges, and the absence of surrounding reaction.

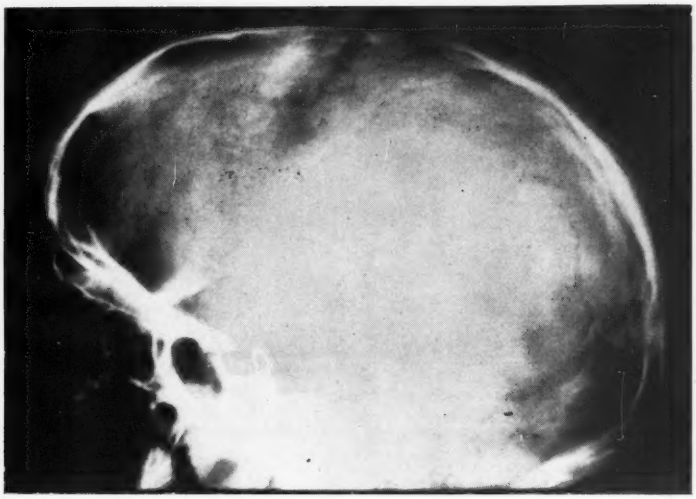

Fig. 112.—Lateral view of initial lesion.

CASE REPORT

R. B., aged 15 years, apprentice gear grinder, was referred on Feb. 22, 1944, on account of a painless swelling over the right side of the forehead of three weeks' duration. There was no other complaint and no history of any serious illnesses, but he had fallen five years previously and had sustained a small wound of the right brow, which had healed readily.

Examination showed a very tall boy, 5 ft. 10 in. in height, of good physical development and average nutrition. He appeared a little pale and the pupils were slightly dilated. There was a prominence over the right frontal eminence, 1½ in. in diameter, above but apparently unrelated to the scar of a small wound of the outer end of the brow. The swelling was fluctuant, with a firm rim, but without any thickening of the overlying skin, which was freely movable over it.

Radiographic appearances (Feb. 22): There is a circular translucent area of the right frontal bone, 1¼ in. in diameter, with a clearly defined, rather irregular edge producing a characteristic 'geographical' outline. Both tables are equally absorbed and there is no surrounding sclerosis. (*Figs.* 111, 112.)

The cystic area was aspirated and the removal of 2 ml. of blood-tinged serous fluid almost reduced the swelling. Microscopical examination of the fluid showed: Large numbers of red cells; large numbers of leucocytes composed of polynuclears 33 per cent, eosinophils 56 per cent, lymphocytes 11 per cent, and large monocytes in moderate numbers. No organisms were seen in Gram-stained films and tubercle bacillus was not found. There was no growth on culture. The appearance suggests that the fluid is from a chronic abscess.

The patient was admitted to hospital for a few days for further observation.

There was no pyrexia. The Wassermann and Kahn reactions of the blood were negative. The visual fields and optic fundi were normal. Exhaustive radiographs of the skeleton showed no other abnormality. The Casoni test for echinococcal infestation was negative. Blood examination showed: Hæmoglobin 87 per cent, red blood cells 5,100,000 per c.mm. Colour index 0·86. Mean size of corpuscle 7·2 m. White cells 9,000 per c.mm., polynuclear neutrophils 72 per cent, lymphocytes 25 per cent, large hyalines 3 per cent. There was a slight degree of poikilocytosis and anisocytosis.

Biochemical estimations: Serum-calcium 10·4 mg. per 100 ml., phosphorus 4·0 mg. per 100 ml., and alkaline phosphatase 13 units. The urine showed no abnormality of type, constituent, or quantity of output.

There was no reappearance of the swelling following aspiration and there was no abnormality of the patient's external appearance on his discharge from hospital. He was observed at intervals for two years, and subsequently he served in His Majesty's Forces. Serial reports over the remainder of the four-year period are:—

Oct. 31, 1944: General condition satisfactory. Frontal region normal in appearance and to palpation. Radiographs show the affected area to be less translucent and to be filling with traces of bone texture (*Figs.* 113, 114).

July 17, 1945: Osteoporosis is now slight, the density being increased by fine shading of thin bone.

Feb. 17, 1948: Serving in the army. Radiographs show the area to be almost completely restored by normal bone of the skull (*Figs.* 115, 116).

Discussion.—The initial diagnosis in this case was Hand-Schüller-Christian disease, and following the examination of the aspirated fluid this was modified to eosinophilic granuloma of bone.

There was no evidence of parathyroid anomaly. The only point of interest arising from the biochemical investigation of the blood was the raised alkaline-phosphatase content of 13 units. As the general state of the patient remained satisfactory and the swelling did not recur after aspiration, biopsy was delayed, and, in the light of persistent improvement as judged by radiographic standards, this was not deemed necessary. Blood-cholesterol estimation was unfortunately omitted.

The gradual spontaneous recovery with restoration of normal bone structure is evident from the serial radiographs taken over a period of four years. In this instance aspiration biopsy proved adequate for the establishment of diagnosis.

FIG. 113.—Appearance eight months later. The area is occupied by traces of newly-forming bone.

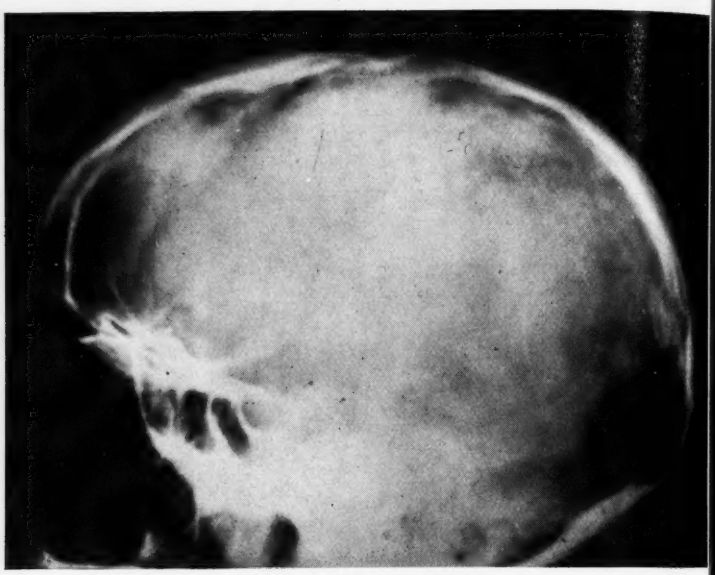

FIG. 114.—Lateral view.

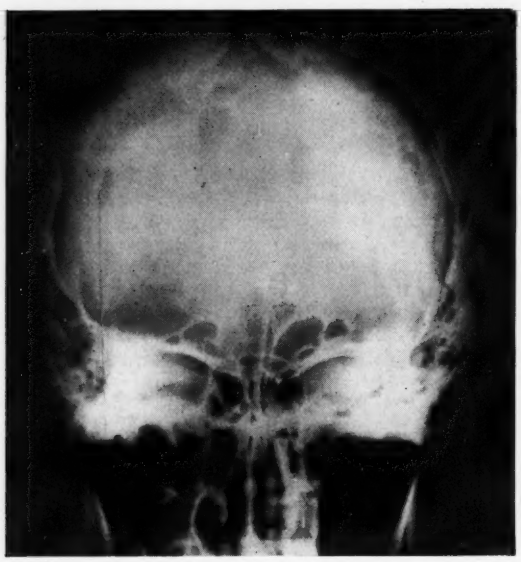

FIG. 115.—Anteroposterior appearance four years after *Figs.* 111, 112. The affected area is almost completely restored by normal bone.

FIG. 116.—Lateral appearance of healed defect.

HISTOLOGY AND AETIOLOGY

Probably the first to report a case of the type under review was Thomas Smith (1865), who found in a child, aged 4½ years, an area of the occipital bone, the site some months previously of a fluctuant swelling, to be occupied by yellow inspissated material and described as a dried-up abscess. Other similar areas were present elsewhere in the skull. Hand (1893) described the triad of features, calvarial osseous defects, exophthalmos, and polyuria, later noted by Kay (1905), and subsequently by Schüller (1915) and Christian (1919), whose names became associated with this syndrome. Schüller reviewed the cases reported up to 1925 under the heading 'Dystosis Hypophysaria'. Later examples were recorded by Kyrkland (1926), Stowe (1927), and Rowland (1928). Finzi (1929) reported a myeloma with prevalence of eosinophils, Mignon (1930) a granulation tumour of bone, and Schairer (1938) an osteomyelitis with eosinophilic reaction. While these cases conformed for the most part to a clinical entity, during the past twenty years the pathological features have assumed a greater significance and there has been a notable tendency to include them in a broader field comprised of such varieties of abnormal lipoid deposition as xanthoma, the simplest form; Niemann-Pick disease, where phosphatid or lecithin is deposited in the liver, spleen, and bone-marrow; Gaucher's disease, wherein a lipoproteid is similarly distributed; Tay-Sachs disease, in which lipoid is accumulated in the nervous tissues; Letterer-Siwe disease (reticulosis, reticulo-endotheliosis, non-lipoid histiocytosis), elaborated by Abt and and Denenholz (1936), which is characterized by widely spread foci of aggregations of histiocytes; and, finally, the Schüller-Christian syndrome, a clinical entity resulting from the localization of the process in the base of the skull.

In 1935 Fraser drew attention to the similarity of the lesions, which he grouped as lipoid granulomatosis, and he described 4 cases involving only the skeleton, the feature common to which was the presence of "a granulomatous tissue which bears certain resemblances to a malignant tumour but which arises as the reaction of reticulo-endothelial tissue to the stimulus of a lipoid which is excessive in amount, and which possibly possesses abnormal characteristics".

Otani and Ehrlich (1940) applied to their case the term 'solitary granuloma of bone'. Jaffe and Lichtenstein carried the review further in detail, and added their designation as 'eosinophilic granuloma of bone'. They referred in particular to those cases of solitary (Farber, 1941) or multiple osseous lesions at the stage at which the pathological appearances were characterized by the presence of many eosinophils. This feature had been well illustrated in the sections of one of Fraser's cases, but the point had not received any particular stress. Jaffe and Lichtenstein considered the possibility that the initial histiocytic eosinophilic process may be replaced by the lipogranulomatous process as had been suggested by Green and Farber (1942), Gross and Jacox (1942), Mallory (1942), Wallgren (1940), Glanzmann (1940), and Flori and Parenti (1937), and they stated that in at least one of their cases healing of eosinophilic granuloma of bone had occurred by direct resolution without incurring the lipoid stage. They concluded that: "eosinophilic granuloma of bone, Letterer-Siwe disease, and Schüller-Christian disease may be considered as different expressions of the same basic disorder. This disorder apparently represents a peculiar inflammatory reaction to some as yet unknown agent of infection and its individual lesions are characterized cytologically by the presence in them at least at first of large numbers of histiocytes. In eosinophilic granuloma of bone the lesions seem to develop only in the skeleton and often only in a single bone and a distinctive imprint is given by the abundance of eosinophils intermingled with histiocytes. The lesions do not tend to become scarified, collagenized, and lipidized, heal readily after simple curettage even without supplementary irradiation, and may heal by resolution even without any therapeutic intervention whatever".

While the lesions in the soft tissues in Letterer-Siwe disease do not show infiltration by eosinophils, the destructive lesions in the bones do show it, and especially in the early stages the lesions are indistinguishable from granuloma of bone, and in the late stages where there is lipoid deposit such a case would pass into the category of Schüller-Christian disease. Thus Jaffe and Lichtenstein, whose careful and exhaustive studies have been quoted at length, have reversed the conception of genesis postulated by Fraser as a local tissue reaction to hypercholesterræmia, and have given their opinion that lipoid deposition may occur as an eventual stage typified in Schüller-Christian disease, may occur without such a transgression, and further that hypercholesterræmia is not an essential feature of the picture. The latter fact has been confirmed by the recent observations of Hamilton, Barner, Kennedy, and McCort (1946).

The relationship to Letterer-Siwe disease was noted as a possibility in one of the cases recorded by Dundon, Williams, and Laipply (1946), where there was a lesion of the sternum and associated lesions of the left supraclavicular, right axillary, and mediastinal glands. Biopsy of both the sternum and the axillary gland showed an appearance said to be diagnostic of eosinophilic granuloma, the variety of cells contra-indicating a diagnosis of Hodgkin's disease. In one of the cases reported by Hamilton and others, where an enlarged perihilar lymph-node and a palpable supraclavicular gland accompanied a lesion of the femur, biopsy of the osseous lesion showed eosinophilic granuloma, and of the gland fibrosis of indeterminate aetiology. These instances cannot be considered as conclusive evidence of the association. The examples quoted in detail by Jaffe and Lichtenstein and reported by Merritt and Paige (1933), and by Chester (1930) in which widespread lesions of the Letterer-Siwe type appeared to be indistinguishable histologically from the various stages of eosinophilic granuloma, are more suggestive that the diseases have some fundamental association.

The present knowledge of eosiniphil leucocytes is incomplete, but there is reason to suppose that their presence is evidence of reaction to certain stimuli occurring in the blood-stream and locally in some infestations and allergic states, and locally

in such granulomatous conditions as that under consideration affecting bone. The exact rationale of their presence is not clearly understood, but it is suggested that in the latter case their appearance may result from the action of some substance generated locally in the tissues rather than as part of some systemic response. The degree of eosinophilia is rarely much above normal, and occasionally as high as 4–11 per cent (Dundon, Williams, and Laipply, 1946). Bacteriological culture is invariably sterile.

While injury has been noted as a precursor in some cases of solitary lesions, this association cannot be clear in examples of multiple foci. It would appear, therefore, that eosinophilic granuloma of bone is the outcome of a toxin at present unspecified. The similarity of the lesions in Boeck's sarcoidosis and in lymphogranuloma benignum or Schaumann's disease warrants some comparison which will be mentioned in the consideration of the pathological features.

TERMINOLOGY

Thannhauser (1947) has recently criticized the use of the term 'eosinophilic granuloma of bone', and he has advised as an alternative to the term 'the essential xanthomatosis of normocholesteræmic type', as suggested in a previous publication (Thannhauser and Magendrantz, 1937), the designation 'eosinophilic xanthomatous granuloma' as being more appropriate, even though it does not embrace the reticulo-histiocytic proliferation of the early lesion.

Consideration of the points already presented in the review of the aetiology leads to the suggestion that a still more appropriate term indicative of the lesion would be 'non-specific granuloma of bone'. This wording has the advantage of simplicity and width of application sufficient to cover the various stages without at the same time implying a specific origin, at present undetermined, or a particular relationship to the systemic diseases mentioned on which pathological opinion is not wholly agreed. For the purpose of clarity, and in order to avoid ambiguity, the term 'eosinophilic granuloma' will be retained in this publication.

INCIDENCE

The most recent and comprehensive contribution by Dundon, Williams, and Laipply (1946) includes an analysis of 48 'acceptable' cases collected from the literature and 5 examples of their own. A contemporary record of 9 cases has been added by Hamilton, Barner, Kennedy, and McCort (1946), and further single examples have been published by Wellensiek (1946), Bakody (1946), Ray and Kellner (1946), Mercer (1945), Riley (1946), and Lucien Leger, Ducroquet, Gauthier-Villars, and Tchakoff (1948). To these the 4 cases described by Fraser may be added to comprise a total of 72 which conform to the type. This number does not include the sixteen examples of the lipoid group recorded in earlier works by Smith, Kay, Thompson, Keegan and Dunn, Grosh and Stifel, Denzer, Hausman and Bromberg, Kyrklund, Rowland, Stowe, or those of Hand, Schüller, and Christian already mentioned and all quoted by Fraser.

Age and Sex.—Skeletal granuloma has been noted especially in children and adolescents, but many cases have been recorded in the older age groups. The 10 cases of Green and Farber (1942) were all under 7 years.

Dundon and others found the ages in 53 cases to be 6 months to 58 years, of whom 34 were under 20 years, and 20 were less than 10 years of age. The ages of the further 20 cases collected which include 9 of a selective group of soldiers (Hamilton and others), and the personal example recorded above, range from 8 months to 32 years.

There seems to be a marked preponderance of males. Dundon and others report the sex ratio as 5 males to 1 female in 43 of their cases of known sex. Of the additional 20 cases 3 were females. Therefore of the total of 63 cases of known sex in 73 examples, 53 were males and 10 were females, thus maintaining the ratio of 5·3 males to 1 female.

LOCALIZATION

Eosinophilic granuloma is found more frequently as a single lesion. Dundon and others recorded 36 solitary foci in 46 of their series in which the site had been noted. It is stated to occur in any bone proximal to the wrist and the ankle. Indeed, in a careful scrutiny of the literature it has not been possible to trace a single case in which the bones of the hand or wrist have been involved. The membrane bones are said to be the most frequently affected. While this is true with regard to the skull and the ribs, the figures relating to the clavicle, mandible, pelvis, and scapula indicate no special predilection for these sites. On the contrary, the femur is found to be affected more often as a solitary site than any other bone excepting the skull. Conversely when there are multiple lesions the membrane bones are the more frequently involved. Green and Farber (1942) record one case in which there were 25 lesions.

Dundon and others have indicated the site in 46 cases in which localization has been accurately noted, and their series has been augmented by a collection of 20 further cases.

In one of their examples of a 'solitary' lesion the sternum was involved together with groups of glands. This case has, therefore, been transferred to the group of 'multiple' foci. The resulting figures are tabulated. A study of this table shows that the total of 66 cases in which the site has been clearly stated includes 47 (71·2 per cent) solitary lesions, and 19 (28·8 per cent) examples in which there were multiple lesions. Of the cases with solitary foci, the skull was involved in 42·6 per cent, the ribs in 15 per cent, and the femur in 20 per cent. The 19 cases with multiple lesions, 108 in all, showed affection of the skull in 16 cases (84·2 per cent); the ribs in 25 instances, and the vertebræ in 10 cases. In 30·3 per cent of the whole series the skull alone was involved.

It may be summarized that eosinophilic granuloma of bone is manifest as a solitary focus in 71·2 per cent, and as multiple lesions in 28·8 per cent of cases. The skull is affected in 42·6 per cent of cases with a single lesion, and 84·2 per cent of cases of multiple lesions. While there is no predilection of the solitary lesion for the other membrane bones,

when the foci are multiple the sites affected are in order of frequency: ribs, skull, femur, vertebræ, pelvis, mandible, humerus.

Table I.—Localization of Lesions in 66 Cases

	Single (71·2 Per Cent)				Multiple (28·8 Per Cent)		
	Dundon and Others 35	Additional Series 12	Total 47	Per Cent	Dundon and Others 11	Additional Series 8	Total 19
Skull	13	7	20	42·6	8	8	16
Femur	6	2	8	17·0	7	4	11
Ribs	6	1	7	15·0	24*	1	25
Humerus	2	1	3	6·4	7	1	8
Pelvis	2	0	2	4·3	5	4	9
Tibia	2	0	2	4·3	2	2	4
Clavicle	1	1	2	4·3	0	3	3
Radius	1	0	1	2·1	0	2	2
Scapula	1	0	1	2·1	2	2	4
Mandible	1	0	1	2·1	4	5	9
Sternum	0	0	0	0·0	1	0	1
Fibula	0	0	0	0·0	2	0	2
Vertebræ	0	0	0	0·0	9	1	10
Face	0	0	0	0·0	2	0	2
Glands	0	0	0	0·0	1	1	2
	35	12	47		74	34	108

* This figure is presumed to include 21 instances of involvement of the ribs in 6 cases with multiple lesions recorded by Green and Farber.

CLINICAL FEATURES

The site and the multiplicity of the lesions and the degree of involvement of the soft tissues determine the symptomatology, which may thus vary from a single local swelling with or without pain to degrees of constitutional disturbance, from entire absence to mild fever, lack of development, and marked malnutrition. Mechanical effects on the local structures may be evidenced by loosening of the teeth, motor disabilities, or by such involvements of the extra-osseous tissues as may be seen in the extreme degree in the Schüller-Christian triad, osseous defects, exophthalmos, polyuria, and polydipsia. Nerve palsies have been recorded, but the literature shows only four examples of pathological fracture. In cases of multiple lesion some sites may be silent, some may be masked by the major lesion, while others may become manifest at a later stage.

Systemic disturbances may be initiated by mild fever and malaise, but the local manifestations of swelling and pain are the more frequent findings. Headache may be present when the calvarium is affected. Osborne, Freis, and Levin (1944) recorded facial nerve paralysis, while diplopia was noted in one of the cases of Hamilton and others. Fraser records a degree of underdevelopment of his cases which exhibited other signs of involvement of the pituitary gland—namely, polyuria and polydipsia. Diabetes insipidus has been reported by Thoma (1943), whose case showed eosinophilic granuloma of the mandible and of other bones, but no evidence of involvement of the base of the skull, and by Versiani, Figueiro, and Junqueira (1944), in which latter case also there was no basal cranial lesion demonstrable by radiological examination. Hamilton and others noted tremor and numbness of the hand and a history of two attacks of loss of consciousness in a case of granuloma of the parietal bone. In relatively few examples such as those of Solomon and Schwartz (1945) and Kernwein and Queen (1943) has the pain been extreme.

In the long bones eosinophilic granuloma is usually manifest as a swelling which may be situated at any part of the bone, diaphysis, metaphysis, or epiphysis. There may be an overlying soft tissue lesion usually associated with erosion of the cortex of the bone. Spontaneous fracture is unusual, but cases have been recorded by Green and Farber (1942), Versiani, Figueiro, and Junqueira (1944), and Otani and Ehrlich (1940), and Jaffe and Lichtenstein have illustrated this feature in a photomicrograph of a portion of a lesion in a femur. When situated in the neighbourhood of a joint, the lesion may produce pain, disability, and muscular atrophy.

When a swelling is fluctuant, aspiration may produce a content showing a large number of eosinophils irrespective of any change in the systemic blood. This feature is depicted in the case recorded above. It is unusual to find much deviation from the normal appearances of the blood. Occasionally leucocytosis has been found, and eosinophilia may be present up to 4–11 per cent (Dundon and others), Bass (1941), and Horwitz (1943). It is of interest that Gross and Jacox (1942), who analysed 84 cases

of Hand-Christian disease, noted eosinophilia of 3–7 per cent in 13 instances. Culture of the material aspirated or removed at biopsy is invariably sterile. The cholesterol content of the blood is usually normal in contrast to that in other groups of xanthomatous disorder with hypercholesteræmia, while the cholesterol content of the affected tissues in the xanthomatous stage may be ten to twenty times higher than that of normal tissues (Thannhauser and Magendrantz). Increase in the alkaline phosphatase of the blood has been noted in two of the cases of Hamilton and others, one solitary and one with multiple lesions, and in that of Riley, who recorded a fall from an initial reading of 9·2 units to 2·5 units within six months in a case of eosinophilic granuloma of the rib. Healing had occurred at the time of the later estimation. The blood-sedimentation rate is occasionally increased.

RADIOGRAPHIC APPEARANCES

The characteristic appearance of eosinophilic granuloma of bone is an oval or circular translucent area. In the skull the edges are usually crenated and produce the typical ' geographical ' outline suggestive of the coalescence of several smaller lesions. The margin is sharp and well defined, and there is rarely any surrounding sclerosis. Both tables are usually equally involved, and the suture lines do not limit extension. There may be evidence of swelling of the overlying soft tissues. The lesion appears to commence in the marrow and, in the case of the rib, expansion of the cortex produces a fusiform enlargement. In the long bones also the cortex may be eroded and even perforated. In some cases the cystic area may be loculated. Periosteal new-bone formation is usually slight and appears to be confined to the lesions of the long bones.

The radiological appearances of eosinophilic granuloma of bone may thus simulate closely those of a large number of conditions, which includes in particular: osteomyelitis, pyogenic, syphilitic, and tuberculous; osteitis fibrosa cystica; osteoclastoma; myelomatosis; Ewing's tumour; osteogenic sarcoma; and osseous metastases; which will be mentioned in the discussion of the differential diagnosis.

PATHOLOGY

The osseous lesions consist essentially of a cystic area, usually in the central part of the bone. This is occupied by soft, hæmorrhagic, or brownish granulation tissue which may be streaked with yellow necrotic material. The supernatant fluid in the softer lesions may be clear or turbid, and microscopical study of aspirated fluid may show abundant eosinophil leucocytes. The histology of sections of the lesion shows sheet-like collections of large phagocytic cells of the histiocyte type interspersed among which there may be conspicuous numbers of eosinophilic cells. Multinuclear giant cells are especially frequent in the areas of hæmorrhage and necrosis.

The characteristic component is a large mononuclear cell derived from the reticulum cell of the marrow. The nucleus is usually single, but some of these cells may contain two or more nuclei. The latter are large, round, and sometimes oval. The cytoplasm frequently contains disintegrating erythrocytes and granular cells, usually eosinophils, and many of the cells show fat droplets. Jaffe and Lichtenstein state that these droplets are not as a rule doubly refractile and probably represent phagocytosed particles of fat naturally present. This feature is also seen in the large giant cells which are of the syncitial multinuclear type. Fraser has noted that the lipoid tends to collect in the perinuclear area and that these ' foam ' cells cease to show any sign of nuclear division. Further, he quotes Sosman (1932), who indicated that the more mature type of foam cell was to be found at the periphery while the smaller mononuclear cell tended to occupy the central region. The endothelial cells of the capillaries are swollen and proliferating, and the recticulum cells of the adventitia may be traced through various stages to the cell described as characteristic. Around a central area of capillary obstruction a concentration of lymphocytes and eosinophil leucocytes may be evident. The eosinophils present irregular or bilobed nuclei, though they may appear small and uninuclear and may so resemble eosinophil myelocytes. Plasma cells and neutrophil luecocytes are also present. Later stages of the process reveal an increase in the numbers of the lipophagic cells and finally of fibroblasts as restitution to the normal occurs.

Jaffe and Lichtenstein have described in detail two specimens of affected rib, in a case recorded by Thurm (1942), in which the evidence of a lipogranulomatous stage was so slender as to lead to the supposition that natural healing may have occurred without the appearance of any greater number of foam cells than are customarily found in chronic pyogenic osteomyelitis. These observers therefore suggested that the intermediate lipogranulomatous stage may not have occurred in the process of resolution in these instances.

Holm, Teilum, and Christensen (1944) summarized the stages of the pathology as: (1) Proliferative with histiocytes and eosinophils; (2) Granulomatous, with increase in blood-vessels and fibrils, reticular cells, giant cells (Touton cells), and incipient lipid phagocytosis; (3) Xanthomatous stage with nests and isolated foam cells; (4) Fibrous stage, which may be considered as a healing phase.

In the absence of definite information regarding a specific origin of eosinophilic granuloma of bone, it is of interest to compare it with such others of the reticuloses as sarcoidosis, lymphogranulomatosis benigna (Schaumann, 1936), and Hodgkin's disease. Some of these conditions have some features in common—namely, the involvement of the reticuloendothelial system and the nature of the reaction invoked by the corresponding metabolite. The latter is essentially a reticular hyperplasia involving the macrophage group of cells, the large mononuclear cell, be its origin the blood, the endothelial cell, or a histiocyte from the tissue and perivascular spaces, giant cells variously typified as Touton, ' mirror image ', Sternberg, etc., and eosinophils. There is also a similarity in the general pattern of localization of the lesions. Hadfield and Garrod (1943) make no reference to granuloma of bone as such in their classification of the reticuloses, but the non-specific origin and the local reaction and

behaviour of eosinophilic granuloma of bone would appear adequate to justify its inclusion at a stage between primary histiocytic reticulosis and the metabolic group of lipoidoses in that it may embody the features of each.

DIAGNOSIS

The appearance on radiological examination of an area of osteoporosis with a well-circumscribed margin and without much surrounding change in the texture of the bone will invoke the possibility of the presence of non-specific (eosinophilic) granuloma in addition to the question of the occurrence of such other lesions as are commonly seen. The importance of accurate differentiation is obvious. The appearances which this lesion may produce in the skull may simulate closely meningioma and epidermoid cysts. The former tends to affect the inner table to a greater extent and the latter causes expansion of both tables, while by contrast the geographical outline of the granuloma is distinctive.

Low-grade pyogenic osteomyelitis may be confusing, but in the chronic case there is a greater degree of density at the periphery. Syphilitic lesions at certain stages may be difficult to dissociate, but the serological reactions provide decisive information. Tuberculosis, especially in the cystic form, may produce a similar appearance only to be distinguished by biopsy, culture, and animal inoculation. Ewing's tumour usually produces a more severe general reaction, and the radiological appearance of typical ' onion peel ' formation at the periphery is a distinguishing feature. Osteitis fibrosa cystica may be confusing, especially when the lesions are multiple, and the diagnosis may have to depend on the result of biopsy and estimation of calcium content of the blood and urine. Osteoclastoma tends to present a clearer reticulum within the cyst. Myelomatosis may be distinguished by the radiological appearances and by the proteinuria when the latter is present. Finally, and of particular importance, osteogenic sarcoma and metastatic tumours may be differentiated, perhaps by radiology and certainly by biopsy, from the solitary and multiple granuloma respectively.

The blood chemistry has no marked feature of distinction, but may provide additional evidence. Biopsy should be carried out in all cases, and histological evidence is of paramount value in the establishment of an accurate diagnosis.

COURSE AND TREATMENT

In the past, treatment has been largely empirical on a somewhat vague basis. In most of the cases reported, radiotherapy has been employed either following biopsy or curettage for the single lesion, or for all affected areas after one initial biopsy. Other cases have been submitted to curettage alone or, in the case of the ribs, to excision, and spontaneous healing has followed in natural course. In some instances distant lesions have become manifest subsequently and have recovered spontaneously. Resolution and healing, which, by radiological standards, is sometimes complete or may be incomplete to the extent of a slight translucence of the previous porotic area, appears to occur within six to twelve months.

Dundon and others state that no death from or attributable to the disease has been reported, but the records show several examples of death from such intercurrent infections as bronchopneumonia, which occurred in *Case* 2 of their series, and in other instances (Fraser ; Merritt and Paige). It is perhaps significant that these cases have shown widespread or extensive lesions. Hamilton and others state that in their experience, while irradiation served to relieve pain, restoration to normal was not appreciably earlier or to a greater degree in the irradiated lesions than in those untreated.

In general there has been little eventual disability. Versiani and others have shown that in the event of pathological fracture of a long bone, such as the femur, there is good capacity for sound healing. Green and Farber found some residual deformity in cases in which the vertebræ had been involved.

Many authors, including Gross and Jacox, have indicated the necessity for a guarded prognosis in cases with multiple lesions, especially affecting the viscera. These latter cases approach more nearly the Letterer-Siwe type, in which the outcome is not favourable. None the less, a review of the recorded cases of non-specific (eosinophilic) granuloma of bone leads to the conclusion that the prognosis is good, and that spontaneous healing is the rule in the course of months. It would not appear necessary, therefore, to employ radiotherapy as a routine procedure, but to take such steps as may be necessary in the particular case to ensure protection of the affected area until healing is adequate for safe resumption of function.

SUMMARY

1. The terminology is discussed of the lesion hitherto described by various names and in particular as ' eosinophilic granuloma of bone '. At the risk of oversimplification the term ' non-specific granuloma of bone ' is suggested, of which the eosinophilic, xanthomatous, or lipoid stages represent merely certain phases of the comprehensive group.

2. The various features of the condition are described and the relation of the osseous lesions to a wider group of systemic disturbances is discussed. It is suggested that non-specific granuloma of bone may be classified as one of the reticuloses.

3. A case is presented of non-specific (eosinophilic) granuloma of the skull which illustrates in particular the capacity of these lesions to undergo spontaneous restitution to the normal structure.

I would acknowledge my thanks to Dr. S. Wray for the pathological details of the case reported ; and to Dr. N. G. Meade and Dr. B. S. Mannheimer for the radiological reports. It is a pleasure also to note my appreciation of the helpful advice accorded to me by my colleagues Dr. J. Steven Faulds and Dr. W. G. Scott-Harden.

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ARTERIOVENOUS ANEURYSM OF THE GREAT VEIN OF GALEN

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WE have been able to find reports of only six cases of arteriovenous aneurysm of the great vein of Galen (Russell and Nevin, 1940; Alpers and Forster, 1945; Jaeger and Forbes, 1946; Lumsden, 1947; Oscherwitz and Davidoff, 1947), only 2 of which were in adults. The following is an account of an additional case, in which the shadow of the lesion and the clinical signs led to a diagnosis of an aneurysm in the region of the colliculi.

CASE REPORT

HISTORY.—A slightly-built woman of 21 was admitted on Jan. 2, 1948, complaining of intense, sharp, stabbing frontal headache radiating to the vertex and occiput. Attacks had occurred at intervals of two to three days during fifteen years and lasted a few minutes. At the age of 2 she had infantile paralysis, a year later a fit, and two more fits when 4 years old. At the age of 8 she had a mild head injury. At 11 "her legs began giving way under her" and she had another fit. During a fit "the eyes turned round and both arms and legs twitched". Consciousness was not lost, urine not voided, and the tongue not bitten. At this time the only abnormal physical finding in a child of normal intelligence and good general condition was slight dragging of the left leg, the tendon-jerks of which were slightly increased, and the plantar response extensor (Dr. A. G. Watkins).

Since this time she had suffered a great deal from headache, generally precipitated by an emotional upset, and her mother noticed that "she was weak in the ankles and fell about a lot". In August, 1947, when she was 20, she fell down a ladder and was unconscious for about an hour. Some hours after regaining consciousness she became unconscious again for a brief time. Since this accident she had "appeared stupid". Though she would answer questions accurately, she lacked concentration and was easily distracted. She was listless and drowsy during the day, and slept well at night. The mother noticed that "when looking up she had to throw her head back to see properly".

On Dec. 18, 1947, she suddenly became ill, with severe headache and vomiting, and was admitted to hospital with a diagnosis of acute encephalitis. She was transferred as a suspect case of intracranial tumour on Jan. 2, 1948.

ON EXAMINATION.—T. 97°; P. 80; R. 22. Tongue dry and furred, breath offensive. Drowsy, rather inaccessible, and suffering from intense photophobia, she could be roused to answer simple questions but her

attention could not be maintained. There was ptosis of the left lid. The left pupil was widely dilated and inactive to light and accommodation. The movements of the eyes were not conjugate. There was external strabismus and loss of vertical movement both upwards and downwards. Both disks were swollen, the left particularly on the nasal side, but no hæmorrhages were seen. X-ray examination of the skull showed a roughly spherical area of 'egg-shell' calcification in the midline in the parieto-occipital region and multiple soft opacities in the frontal region.

On Jan. 9, ventriculography demonstrated deformity of the aqueduct of Sylvius, with dilatation of the third and lateral ventricles (*Fig.* 117). Samples of ventricular fluid were normal.

FIG. 117.—Ventriculogram showing spherical area of calcification, gross dilatation of the ventricles, and deformity of the aqueduct.

OPERATION (Jan. 9, 1948).—Torkildsen's operation was performed, the right lateral ventricle being drained into the cisterna magna by means of a small rubber tube. The tumour was regarded as an aneurysm and not explored.

PROGRESS.—She was pyrexial for four days following operation. Examination of the C.S.F. on Jan. 14 showed a faintly xanthochromic sterile fluid. During the next ten days there was a sustained improvement. The headache was relieved, and she became more accessible, and it was possible to converse with her. On the seventeenth post-operative day the temperature suddenly rose. The lateral ventricles were tapped and the pressure found to be equal and normal, but the fluid was faintly xanthochromic and turbid, and despite penicillin therapy she died three days later. Necropsy was performed.

AT AUTOPSY.—An ovoid varix measuring 3 cm. by 2·4 cm. was found attached to the lower border of the falx cerebri at its junction with the tentorium cerebelli (*Fig.* 118). It extended forwards and downwards to a point 1·3 cm. below the incisura of the tentorium, which, with the adjacent parts, was adherent to it and partially separated it from the posterior ends of the cingulate gyri. It pressed upon both the tectum of the midbrain and the superior vermis of the cerebellum. The wall of the varix varied from 0·5 mm. to 1·0 mm. in thickness and had the histological structure of a vein, with calcification of the media, most marked in the inferior region. Its cavity extended backwards into the falx cerebri for a distance of 1 cm., and in this region it was traversed by several short fibrous bands. It contained a post-mortem blood-clot. Both the inferior longitudinal sinus and the straight sinus entered the varix at points 1·5 cm. apart (*Fig.* 119). Anterosuperiorly four small blood-vessels, two on each side of the midbrain, extended forwards from the sac. Those on the right side included a thick-walled vessel 1·5 mm. in diameter, which proved histologically to be an artery. This vessel branched and anastomosed with a neighbouring vein to form a small plexus.

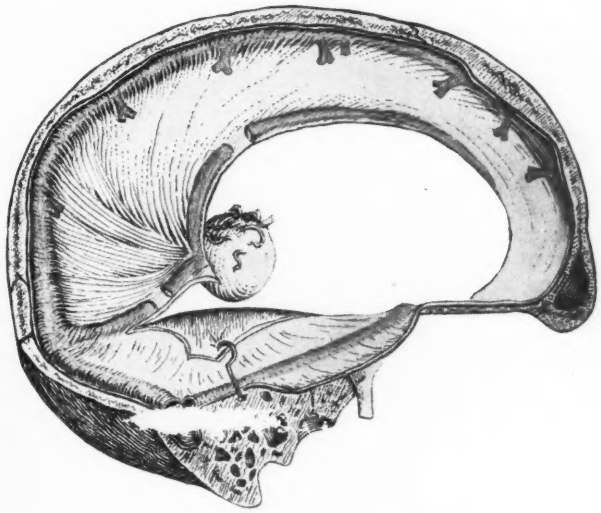

FIG. 118.—Diagram illustrating the position of the aneurysm.

Both lateral ventricles showed considerable dilatation, the convolutions over the vertex were flattened, and there was a well-marked cerebellar pressure cone. There was no evidence of infection.

FIG. 119.—Photograph of aneurysm.

Several small osteophytic outgrowths measuring up to 1·0 cm. in diameter were present over the inner surface of the frontal bone, being adherent to and apparently extending into, the dura mater.

COMMENT

Arteriovenous aneurysm of the great vein of Galen has been reported in two male infants by Russell and Nevin and by Lumsden in a male of 7 months. In these the big head, the wide open fontanelles, progressive dilatation of the veins of the face, scalp, and neck, headaches, and fits were noted. Lumsden records coarse nystagmus, intermittent strabismus, and small contracted pupils inactive to light. In a boy aged 4½ (Jaeger and Forbes, 1946) bilateral arteriovenous communication of the posterior and middle cerebral arteries was associated with

dilatation of the great vein of Galen and enlargement of the venous sinuses. This child had been observed since birth. Delivery had been difficult because of an abnormally large head. The early symptom was repeated severe epistaxis. Prominent veins of the face and scalp were noted, and these became smaller after an attack. These attacks gradually ceased after the age of 3 and were replaced by intermittent headache and vomiting, recurring at intervals of several days to three weeks. When 4 years of age the headache was of almost daily occurrence, his gait was unsteady, and he began to deteriorate rapidly. Within two months he was unable to walk, his speech had become unintelligible, and an alternating squint had developed. At autopsy the vascular abnormality was found to be associated with dilatation of the ventricular system above the aqueduct. In both our case and in a youth of 18 (Alpers and Forster, 1945) there were histories of frequent severe frontal headaches during a period of years, but signs and symptoms of an intracranial space-occupying lesion were lacking until three months before death. It is noteworthy, however, that a symptom indicative of loss of vertical movements of the eyes was noticed by the mother of our patient four months before her admission to hospital, and that later localizing signs referrable to the quadrigeminal plate were elicited. In contrast, the only complaints of a woman of 27 who presented no abnormal neurological signs were attacks of headache during nine months and intermittent blurring of vision during three months (Oscherwitz and Davidoff, 1947). The calcified wall of the aneurysm was demonstrated radiologically in all these adults. Hydrocephalus, which was present in every case except that reported by Oscherwitz and Davidoff, would appear to be due to distortion of, and pressure upon, the aqueduct.

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A NEW METHOD OF VENOGRAPHY WITH PARTICULAR REFERENCE TO ITS USE IN VARICOSE VEINS

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Two methods of venography have been described: by intra-arterial injection of the contrast medium and subsequent radiography of the veins, which appears to have little if any use except within the skull; and by intravenous injection and radiography of a moving column of blood and contrast medium.

The difficulties in the interpretation of the films, particularly those of the calf veins, have been noted before (Bauer, 1941; Allen, 1938, who found the result poor in one-third of his cases due to artefacts) and are due to incomplete filling of the veins with contrast medium, this in turn being due to the fact that organic iodides are heavier than blood (Kellberg, 1943) and, in the slowly moving column, tend to sink to the bottom of the vein, as can be seen in lateral views (*Fig.* 125), and because the medium is continuously diluted by fresh blood coming into the limb. Indeed it is only possible to obtain good films by surgically exposing the vein and injecting the material rapidly.

It is to be remarked also that in previous reports seldom, if ever, are pictures of the femoral veins obtained from the injection in the foot (*see* particularly the paper by Dougherty and Homans, 1940) and never of the iliac veins or inferior vena cava. To obtain pictures of these the saphenous vein is injected—which of course entailed exposing it surgically.

The actual techniques differ but little and are well described by Hellsten (1942). Briefly they consist of entering a vein, usually by a 'cut-down' for the lower limbs, injecting the opaque medium as quickly as possible, and taking films at stated intervals during and after the injection. The vessels used for the injection are generally behind the external malleolus for the calf (with sometimes a tourniquet on the ankle to prevent filling of the superficial veins), the long saphenous at the knee, or higher, for the thigh or iliac vessels, and at the elbow for the arm and subclavian veins.

As these methods have given variable results in the past and as in practice they are troublesome, unreliable, and only show a small part of the lower limbs, I decided to try the effect of cutting off all blood-flow in the limb with a view to pooling the contrast medium in the end veins and then washing it on as a bolus. To my surprise the contrast medium did not remain in the foot and lower calf area, but filled all the leg veins, the popliteal, and sometimes half the femoral vein. Although the medium was much more diffused than expected, it, nevertheless, remained in fairly high concentration and the obvious step was next to release the tourniquet and X-ray the upper femoral and iliac veins soon after release; upon which good filling of the femoral and sometimes of part of the iliac was obtained but it was obvious that the medium was too dilute in the common iliac vein and inferior vena cava to give any pictures.

I, therefore, prevented any dilution by blood from the opposite limb by placing a tourniquet around the thigh of that limb, and attempted to slow the venous return from the pelvis and limb injected by a forcible expiration just before the release of the tourniquet on the side being X-rayed; and this always gave a good film of the iliac veins and sometimes of half or more of the inferior vena cava itself.

TECHNIQUE

For the Lower Limbs.—It is better to vary the method a little depending on whether the iliac and upper femoral veins (*Fig.* 120) or the calf veins (*Figs.* 121, 122) need to be chiefly outlined.

For the *iliac and upper femoral veins* the patient is positioned over the Potter-Bucky apparatus. He is then sat up with his legs dangling over the side of the couch and a sphygmomanometer cuff put well up on the thigh of the affected side; another tourniquet is placed on the opposite thigh and fixed tight enough to prevent venous return from that limb, and a rubber tourniquet is put on the ankle of the affected side tight enough only to occlude the superficial veins.

FIG. 120.—Venogram of a normal patient. This film was taken 7 seconds after the release of the thigh tourniquet with a 1-second exposure. It is not unusual to find some part of the superficial femoral vein to be double. There is slight reflux of blood into the profunda femoris, which is rare, and into the internal iliac vein, which occurs occasionally.

When the veins on the foot of the affected side are standing out, a No. 16 hypodermic needle is inserted into any one of them and pushed well in. It is best to do this with the needle unattached to the syringe. The patient then lies down, the sphygmomanometer cuff is inflated to above the systolic blood-pressure and 30 c.c. of 35 per cent pyelosil are injected (which takes about 40 seconds). The syringe is detached, but the needle is left in place—this prevents hæmatoma formation—the patient breathes in and then out forcibly and holds his breath. The pressure in the sphygmomanometer cuff is rapidly released and the film taken 5 seconds after this with a 2-second exposure.

The exposure for the pelvic films allows a good deal of variation. While the best time seems to be 5 seconds after the release of the thigh tourniquet with a 2-second exposure, a much longer exposure works quite well as the medium is present for some time in the slowly moving stream. Thus, an 8-second exposure as soon as the tourniquet is released; or wait 4 seconds and then give a 3-second exposure etc.—the optimum time seems to be within 8 seconds of release of the pressure in the sphygmomanometer cuff for a normal patient.

FIG. 121.—Venogram of a normal patient. This film shows the venæ comites of the posterior tibial and peroneal veins with frequent bulgings at the valves—the usual appearance in the leg. The anterior tibial vein is not filled—the normal finding. There is poor filling of the beginning of the popliteal vein (*see* text). The popliteal vein shows the valves commonly found about the level of the knee-joint. The muscle veins are hardly seen, which is, again, normal in the anteroposterior view of the leg—that is, in the first film. Note that none of the superficial veins are filled. With the exception of the beginning of the small saphenous vein, none fill in a normal person when a tourniquet is used at the ankle.

Obviously a box or other method for multiple exposures, particularly with obstruction of the inferior vena cava where the time of flow is slowed, would be better, but this is rarely available in most centres.

For the *calf veins and the superficial femoral*, the procedure is as before except that the sphygmomanometer is inflated to just over the diastolic pressure (in practice about 140 mm. of mercury works very well in all cases) which allows a certain amount of blood into the limb and produces a better spread of the opaque medium and better outline of the vessels. When the injection of pyelosil is completed, an anteroposterior film is taken of the leg, from the ankle to near the knee, with the

foot turned in 45° to get the maximum space between the bones; the patient is then turned on to his side and a lateral film is taken of the same area, followed by a lateral one of the upper calf and thigh up to the tourniquet; finally the patient is turned on to his back again and an anteroposterior film is taken of the upper calf and thigh.

It is generally found that subsequent films of the pelvis do not show the veins because the medium has become too dilute before the release of the sphygmomanometer cuff.

This technique is especially indicated where varicose veins are being investigated. If the varicose veins are severe, 40 c.c. of medium are needed because there is so much blood in the dilated veins that the pyelosil is considerably diluted.

FIG. 122.—Venogram of a normal patient. These films are taken with the patient turned on to his side and are the second and third exposures (*see* text). The anterior tibial veins are filled—which is most unusual. The posterior tibials are those standing well away from the bones. The peroneal veins are partially hidden by the shadow of the bones and by the posterior tibial veins. The muscular veins are in three main groups, which is usual but not constant. The lower group arises from the posterior tibial veins; the upper two probably from the peroneal, although it is difficult to be sure. These veins appear to be without valves. The popliteal vein is formed quite high, which is not uncommon, and from it the small saphenous vein is partially filled, which is also not uncommon. The superficial femoral vein shows the increased density at the site of valves in its lower third.

Fig. 123. *Fig.* 124.

FIG. 123.—Venogram of a normal patient. This film shows very well the fish-tail appearance of the valves, with increased density above and a lighter shadow below. It is unusual to see small veins filling from the femoral vein.

FIG. 124.—Venogram produced *without* any tourniquet at the ankle. This film shows the utter confusion that can occur when the superficial and deep veins are all filled, particularly, as in this case, in the presence of varicose veins.

It is to be noted that a tourniquet is always put on the ankle of the affected side to prevent filling of the superficial veins from below. For radiographs of the higher veins it is essential to have the opaque medium only in the direct route to the pelvis, and for the leg and thigh veins it is important to see the deep veins well and the communications from the deep to the superficial veins, without the confusion of a network of superficial vessels. That these can be most confusing is seen in *Fig.* 124.

For the Upper Limbs.—This is extremely easy. To view the veins of the arm, a tourniquet is placed high on the arm and any vein used for injection.

To see the axillary, subclavian, innominate vein, and superior vena cava, the technique used for angiocardiography is employed (Robb and Steinberg, 1939)—smaller quantities of the medium, however, are quite sufficient.

DIFFICULTIES AND DANGERS

The veins on the foot are usually small and often free in the subcutaneous tissues and, therefore, may be difficult to puncture. If, however, the limbs are hanging until the veins are well filled and

FIG. 125.—Venogram made *without* the use of a thigh tourniquet—that is, with a moving stream of blood. Note how the contrast medium is sunk to the bottom of the vein—a point noted by Bauer (1942).

a No. 16 needle with a short bevel is used, without a syringe attached, it is not difficult to ender the vein. There is often a vessel on the medial side of the base of the big toe which is easy to enter as it does not slide in the subcutaneous tissues.

The needle must be well into the vein or leaking will occur, but if this does happen only slight discomfort follows.

There have been three cases reported of thrombosis following venography with 50 per cent diodrast (Homans, 1942, one case; Scupham, De Takats, Van Dellin, and Marcus, 1942, two cases). Homans' patient had a strong tendency to thrombosis. No details were given about the other two patients except the strength of the medium used. In my experience 35 per cent pyelosil never gives any discomfort—50 per cent, however, sometimes causes aching in the calf and foot and I therefore no longer use it.

The most severe general reaction which has occurred in about 100 cases is vomiting, and that seldom. Other general reactions do occur but appear to be very rare (Pendergrass, Chamberlin, Godfrey, and Burdick, 1942), but the ocular test for sensitivity (Archer and Harris, 1942) is carried out as a precaution although the value of such tests seems to be doubtful (Robins, 1942). The test consists of putting a drop of the material in the conjunctival sac, closing the eye for one to one and a half minutes and seeing if any redness develops. A mild reaction can be neglected; a moderate or severe one must be regarded. If in doubt ½–1c.c. of pyelosil can be slowly injected intravenously and if no reaction develops in five minutes the main injection may be given. It is wise to have adrenaline to hand in case of sensitivity.

INTERPRETATION OF THE FILMS

1. Normal.—The pelvic veins and inferior vena cava appear as broad smooth channels without any evidence of valves (*Fig.* 120).

The femoral vein also is broad and smooth, but may be double, and commonly shows one or two valves in its lower third (*Figs.* 120, 122, 123). The profunda femoris has not been seen in any case.

The popliteal vein is often very indistinct at its origin, possibly because the anterior tibial does not fill with the medium and the blood from this vein dilutes the pyelosil just at its point of entry into the popliteal, giving a poor shadow. There is commonly a well-marked valve present just above the level of the knee-joint or a little higher and the small saphenous vein is often seen to fill for a short distance when there is no evidence whatever of varicose veins (*Figs.* 121, 122).

Leg Veins.—The anterior tibial rarely fills.

The posterior tibial and peroneal veins are normally double and surprisingly big. They show many valves (*Figs.* 121, 122).

In the lateral view the veins of the calf muscles are large and profuse, and usually in three distinct groups, and appear to be without valves (*Fig.* 122). If the patient is put supine, the femoral artery occluded with the fingers, and a sphygmomanometer cuff on the thigh rapidly pumped up to above the systolic blood-pressure so that no blood enters the limb, and the injection then made, the muscle veins appear as big as ever, showing that this is their normal state and not an artefact from congestion of the limb—admittedly there is some congestion from the blood squeezed out at the site of the tourniquet and from the pyelosil injected, but that cannot be very great.

2. Abnormal.—

Varicose Veins.—There have been numerous reports on the use of venography in varicose veins (Pomeraney and Tunick, 1933; Imber, Beaver, and Sheehan, 1944; Allen, Barker, and Hines, 1947; Massel and Ettinger, 1948; Bauer, 1948; and others).

The technique used has been either to inject the varicose veins and watch the result (Pomeraney and Tunick) or to use the usual technique already described.

Both are fundamentally unsound because one thing which must be known for successful treatment is the site of those varicose veins which fill from the deep vessels. Injecting into the varicosities only shows where they empty; and the usual technique may or may not show the superficial veins (when a tourniquet is used on the ankle), as the outflow from the leg is too swift, and, without the tourniquet on the ankle, one does not know if the varicosity is filled from the deep vessels in the calf or thigh or directly from the superficial foot veins, even when

connexions can be seen between the superficial and deep systems.

If the deep veins are filled from the foot (the tourniquet at the ankle preventing the varicosities filling via the superficial vessels) and the flow arrested by a tourniquet at the groin, then it is sure that any superficial veins outlined are filled from the deep veins due to incompetent valves.

The findings have been interesting, for not only are the connexions between the superficial and deep veins well shown (*Figs.* 126, 127), but also the great size of the deep veins in many cases of superficial varicosities—veritable lakes of blood—the same being true of many cases of varicose ulceration, suggesting that the cause of varicose veins may lie in the deep vessels rather than in the superficial, which is in agreement with the findings of Bauer (1948).

Fig. 126.

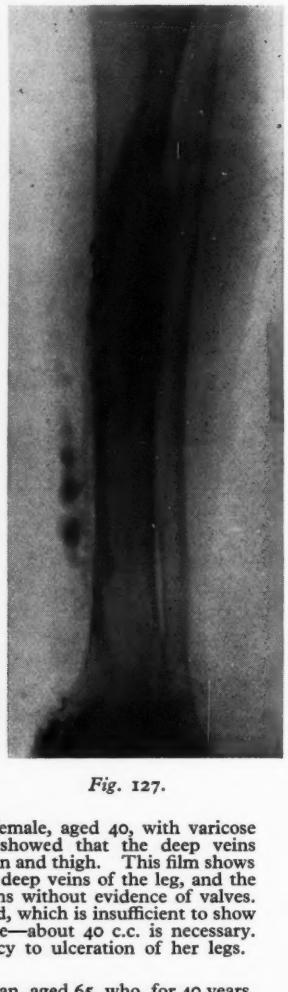

Fig. 127.

FIG. 126.—Venogram of a female, aged 40, with varicose veins. The subsequent films showed that the deep veins were patent in the popliteal region and thigh. This film shows the filling of the varix from the deep veins of the leg, and the large dilated posterior tibial veins without evidence of valves. Only 20 c.c. of pyelosil were used, which is insufficient to show all the veins in this type of case—about 40 c.c. is necessary. The patient showed no tendency to ulceration of her legs.

FIG. 127.—Venogram of a man, aged 65, who, for 40 years, following an injury to the pelvis, had had gradually increasing varicosities of his legs, thighs, and abdominal veins. He now presented as an obvious clinical case of occlusion of the I.V.C. He had had varicose ulcers six years before, but these were now healed. This film shows the filling of the varicosities of the leg from the deep veins of the calf and, as in *Fig.* 126, enormously dilated posterior tibial veins.

VENOGRAPHY IN THROMBOPHLEBITIS OF THE LOWER LIMB

There have been many reports on its use in the diagnosis of thromboses (Veal and McFetridge, 1935; Bauer, 1941; Hellsten, 1942; De Bakey, Schroeder, and Oschner, 1943; and others) but there has been much argument about its value because of the liability of mistaking artefacts for thromboses.

Its use in the diagnosis of doubtful cases is itself doubtful, for even in quite definite clinical thrombophlebitis the films may show nothing abnormal, or perhaps the peroneal veins will not be shown, or only one group of muscle veins be outlined, all of which can occur in a normal person.

On the other hand, in very early cases, the origin of the thrombus in the calf veins can sometimes be seen as 'ghosts' of those veins—that is, the main part of the vein is filled with clot, the pyelosil appearing as a thin ring around the clot.

In more severe cases the extent of the thrombosis can be seen either as a thin outline of the vessels affected, or as a definite block with the pyelosil ascending via accessory channels—information which may be of value if ligation of the vein is contemplated.

This work was done with the assistance of a Beaverbrook Fellowship, which I gratefully acknowledge.

My thanks are also due to the members of the X-ray Department of the Bristol Royal Infirmary, particularly to Sister Sammons, for their patience in much trial and error; to Mr. Croot, F.R.C.S., for permission to study cases from his vein clinic; and to Professor R. Milnes Walker, M.S., F.R.C.S., for his encouragement and advice.

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DUODENAL DIVERTICULA

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HISTORICAL

DUODENAL diverticula have been recognized with increasing frequency since their first description by Chomel (1710). They were recorded by Morgagni (1761), and Fleischman (1815) gave details of a case observed at post-mortem examination. Only isolated cases can have been recorded, as Cruveilhier, writing in 1849, stated that diverticula of the intestinal tract did not occur between the œsophagus and the colon. In the present century they have been recorded more frequently owing to the increasing use and more accurate interpretation of radiographs. Wilkie (1913), Davis (1913), Spriggs and Marxer (1925), Morton (1940), Friedlaender (1937), Tanner and Benians (1939), Ackermann (1943), Whipple (1942), and many others have added to the literature in recent years.

CLASSIFICATION

They are of two main varieties—primary and secondary. Primary diverticula have also been classified as ' false ', as they do not have a complete muscle coat in their entirety, or ' congenital ', to distinguish them from the acquired variety, although there is no general agreement that they are, in fact, present at birth.

Secondary diverticula are always a complication of long-standing duodenal ulcers, and they are therefore definitely ' acquired ', a description which is sometimes applied to them, or ' true ', in that they have a complete covering of all the coats of the intestinal wall. Friedlaender (1937) described the formation of these secondary diverticula following contraction in the region of an old-standing duodenal ulcer, and points out that, owing to the distortion caused, food forced through the pylorus will be directed on to one particular part of the duodenal wall, and that this may gradually pouch out and form a diverticulum. He points out that these pouches only retain food for a few seconds, that they do not constitute a therapeutic problem of themselves, and that treatment is the treatment of the causative ulcer.

The primary type occurs most commonly on the concave side of the duodenum, although cases have been described arising from its front or outer border. Ackerman (1943) suggests that they are abortive attempts to form a supernumerary pancreas. Spriggs and Marxer (1925) point out in support of a congenital origin that they occur in that part of the duodenum which is supported by the head of the pancreas, and which would be the least likely part to pouch out as a result of increased pressure there. In addition they occur in the region of the ampulla where developmental outgrowths normally take place, and where abnormal ones would therefore seem to be likely; and lastly that they may be associated with other congenital abnormalities.

In favour of an acquired origin they point out that they are seen in elderly patients, and they produce evidence that they increase in size. A radiograph in a patient of 50 showed a diverticulum the size of a cherry stone, and eight years later it was 1½ in. across. A second patient of 51 had a diverticulum the size of a pea, and four years later it measured 18 mm. in diameter. The concavity of the duodenum is the part where the muscular wall is penetrated by the blood-vessels, and therefore the site of weak areas where pouching from pressure would be expected. Lastly, they suggest that pressure in the duodenum is likely to be higher than in the rest of the small intestine owing to the closure of the pylorus while peristaltic waves are passing along it. They incline to the view that these diverticula may be acquired, but say they cannot be dogmatic about it.

Spriggs and Marxer (1925) found 38 diverticula in 1000 barium meal examinations, 20 occurring in females and 18 in males; 32 were single and 6 multiple. They found 1 in the first part, 30 in the second part, 16 in the third part, and 4 in the fourth part. Ackermann (1943) made casts of the duodenum to determine their frequency and found that they occurred in 11 of 50 specimens. In 3 cases there were two diverticula. He found 5 in the second part, 1 at the junction of the second and third parts, 5 in the third part, and 3 in the fourth part. They all occurred from the inner border and 7 were buried in the pancreas. Costello (1933) says that in 4 of 6 cases they were not demonstrated until after more than one barium meal. Friedlander (1937), reporting 13 cases, found that 7 were solitary and 6 multiple. He found 1 in the first part, 8 in the second part, 2 in the third part, and 4 in the fourth part. Avancini (1939) says that two-thirds occur in the region of the ampulla.

SYMPTOMS

They may be quite symptomless and may be discovered unexpectedly on barium-meal examination. Davis (1913) thought that in 61 cases only 1 caused symptoms, and Whipple (1942) stated that in 57 cases rather more than half gave rise to symptoms. Odgers (1930) said that the great majority do not cause trouble. Warren (1943) stressed that other causes of symptoms should be ruled out before it is decided that the diverticulum is at fault. Guthrie and Brown (1938) suggest that a large neck allows intestinal contents to enter and that they do not empty as they have no muscular coat. Morton (1940) believes that symptoms may be due to: (1) Mechanical factors such as perforation; (2) Inflammatory reactions; and (3) Carcinoma developing within the diverticulum. Beals (1937) divided the symptoms into: (1) An ulcer type of symptoms; (2) Nausea, vomiting, and upper abdominal pain; (3) Vague minor symptoms. Spriggs and Marxer (1925) found distressing flatulence, fullness, or distension; pain or aching; nausea; vomiting or hæmatemesis and jaundice. Friedlaender points out that inflammatory reactions should be rare, as the

bacterial flora of the duodenum are not so likely to cause trouble as would be the case in the colon. Strode (1945) reported a case which had developed an adenocarcinoma, and which was successfully resected together with the head of the pancreas and the pyloric part of the stomach. Further cases of malignant disease have been reported by Bengolea (1928) and Morrison and Feldman (1926). Melville reported a case with jaundice where there were two diverticula in the second part.

TREATMENT

Lahey (1940), writing on surgical removal, states that from the inner wall they may be difficult to dissect and are often an unjustifiable hazard. Friedlaender (1937) describes 13 cases which were all treated non-operatively. Spriggs and Marxer (1925) favour postural drainage controlled by radiography, lubrication by liquid paraffin, intestinal antiseptics, correction of abnormalities such as achlorhydria, carminatives, and a plain, well-prepared diet which should be well masticated. The earliest deliberate surgical attack was reported by Forsell and Key (1916). In their first case, when the diverticulum was in the second part, it was not removed, and the second case, which was arising in the third part, was exposed through the gastrocolic omentum and excised. Maclean (1927) advised opening the duodenum, inserting a finger into the diverticulum and dissecting it out with the finger in situ, and then ligating its neck. Later writers advocate a lateral approach, mobilizing the second part of the duodenum and approaching the sac through the head of the pancreas from behind. The case reported by Chauvenet (1939) appears to be unique in that the common bile-duct and the pancreatic duct opened into the diverticulum. They were divided and reimplanted into the duodenum, but the patient died three weeks after operation with a biliary and pancreatic fistula. Guthrie and Brown (1938) treated a case by posterior gastro-enterostomy, but six months later they undid the anastomosis and removed the diverticulum as the symptoms had not been relieved.

CASE REPORT

G. W., a railway porter aged 54, attended the Out-patient Department on Nov. 27, 1947, complaining of epigastric pain and increasing loss of appetite for five years. His pain came on in attacks of about three weeks' duration, and occurred about four times a year. It was dull and aching in character and became worse about half an hour after food. He said that he could not eat fatty foods and that he could obtain relief by taking alkalis or a hot drink. During an attack he vomited two to three times a day. The vomitus was small in amount and dark in colour. During this time his appetite had gradually become worse and he had lost 1½ st. in weight. In September, 1947, he had a more severe attack and had done no work since that time.

On Examination.—He was a lightly built man, older looking than his years and with evidence of some arterio-sclerosis. His electrocardiogram was normal. Physical examination showed some general tenderness in the epigastrium, more marked on the right side, but no fullness or tumour. A barium-meal examination (*Fig.* 128) showed a normal œsophagus and stomach, a normal duodenal cap, and a large diverticulum of the second part of the duodenum, which was tender on pressure.

He was admitted to my wards on Jan. 21, 1948. As his symptoms continued, he was anxious to be operated on and said that he had been unable to get relief in spite of having undergone treatment on medical lines.

At Operation.—On Jan. 26 his abdomen was opened through an upper right paramedian incision, and his stomach, liver, and gall-bladder were found to be normal There were no diverticula in the jejunum. An incision was made in the peritoneum to the outer side of the

Fig. 128.—The X-ray appearances, with an air bubble above the barium-filled diverticulum.

second part of the duodenum, which was then mobilized together with the head of the pancreas. No sign of the diverticulum could be seen, but on compressing the upper and lower parts of the mobilized duodenum between the examining fingers and thumbs, air was forced into the diverticulum, which became distended and was seen to be lying in the head of the pancreas, covered by a thin layer of pancreatic tissue posteriorly. A horizontal incision was made from the duodenal border into this tissue and the diverticulum was found to be about 2 in. in diameter and opened into the duodenum by a neck ¾ in. across behind and below the common bile-duct (*Fig.* 129). It was dissected out carefully and divided at its neck. During the dissection the common bile-duct was seen, but the pancreatic duct was not visualized. Only a small amount of bleeding occurred and was easily controlled. The duodenal mucosa was closed by a continuous catgut suture and then buried by a series of interrupted stitches drawing together the muscle layers. The defect in the pancreas was also obliterated by interrupted catgut sutures. The duodenum was allowed to fall back into its original position and the abdomen was closed with a dental rubber drain down to the incision in the peritoneum on the outer side of the duodenum. Continuous gastric suction was employed for three days and the drain was removed after 48 hours. On the second day he had a considerable amount of epigastric

pain which radiated to the right shoulder, but his general condition was good. His temperature rose to 99·6° F. and his pulse was 96. On the fifth day there was a little watery discharge, probably pancreatic in origin, which continued for three days. His wound healed by primary intention and he was discharged from hospital on Feb. 22.

He was seen on June 9, 1948, when he was taking a normal diet, having no indigestion or discomfort of any kind, and he had returned to his employment as a railway porter.

PATHOLOGICAL REPORT.—This showed a diverticulum lined by intestinal mucosa bearing numerous goblet cells. There was a well-defined muscularis mucosæ. Its wall

FIG. 129.—The duodenum has been turned over to the left and the pancreas incised to show the diverticulum.

was otherwise devoid of muscle apart from a few strands near its junction with the duodenum. The diverticulum contained a rolled-up piece of what appeared to be a tomato skin, about 1 in. in length, but it could not be identified with certainty.

DISCUSSION

Although duodenal diverticula are probably present in 2–4 per cent of the general population, only a small proportion of them give rise to symptoms. A complete abdominal examination should be carried out and only when it is negative should the diverticulum be held responsible. The symptoms are so variable that a diagnosis can only be made on the results of radiological examination. They are not likely to be discovered on exploratory laparotomy, as they collapse when the duodenum is manipulated and they are notoriously difficult to find even when a definite pre-operative diagnosis has been made. At operation they can best be demonstrated by compressing the mobilized duodenum between the examining fingers and thumb on the two sides of their suspected site. In this way air is forced into them and they stand out and become obvious. Invagination of the duodenal wall in an attempt to locate the opening into the gut may be tried, but would seem to be a more clumsy method, and deliberately opening the duodenal in order to see the opening is not advocated. Before surgical treatment is advised, patients should be treated on medical lines as suggested by Spriggs and Marxer (1925), and it is only in those cases which do not respond that removal of the diverticulum should be advised. Indirect attacks on the condition, as by short-circuiting operations, should not be attempted, and if an operation is thought to be justified, it should aim at the removal of the diverticulum. Careful dissection of the diverticulum to avoid injury to the common-bile duct, the pancreatic duct, and the pancreatico-duodenal artery, and careful closure of the defect in the duodenal wall, should make the operation a reasonably safe one. This closure is in a part of the duodenum which is devoid of peritoneum and it may be reinforced by drawing across a piece of the great omentum and fixing it to the suture line. A small drain of soft collapsible rubber is an additional safeguard to the patient, and a source of comfort to the surgeon.

The problem as to whether these diverticula are present from birth has not been solved, but they are undoubtedly recognized with greater frequency in the older age groups. It is certain that they increase in size from the time of their formation, and that they only attain proportions sufficient to cause symptoms in later life. At this time their contents may not empty and so stagnation and decomposition can take place. This may be aggravated by the entrance of foreign bodies or indigestible articles taken by mouth, as in the case reported here.

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RUPTURE OF A SPLENIC ARTERIAL ANEURYSM AS A FATAL COMPLICATION OF PREGNANCY

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RUPTURE of a splenic arterial aneurysm is an extremely rare cause of death in pregnancy although various types of intra-abdominal hæmorrhage have been described from time to time. Barber (1909) recorded a case in which intra-abdominal hæmorrhage had occurred " probably from some vein in the pelvis ". Potocki (1918) described a fatal case where he considered hæmorrhage to be due to tuberculosis of the left kidney, which had been responsible for rupture of a branch of the renal artery, probably with prior formation of a miliary aneurysm. Latzko and Schiffmann (1928) suggested that bleeding in the region of the kidney might arise in nephritis, hæmophilia, neoplasm, etc., or spontaneously from capillary oozing. Bruce (1937) observed that severe spontaneous intra-abdominal hæmorrhage may arise from ruptured ovarian cyst, from the veins crossing over the surface of a large fibroid, from rupture of the spleen or liver, or from rupture of the splenic vessels in hepatic cirrhosis, as well as in arteriosclerosis. Lawen (1939) described 2 cases, in the first of which hæmorrhage arose from rupture of the vena cava which was the result of phlebitis dissecans, whilst in the second a sarcoma of the lumbodorsal fascia was associated with a cystic hæmatoma in the kidney region. Kenny and Doniach (1945) suggested that congenital aneurysm or rupture of uterine or pelvic veins might give rise to hæmorrhage, and in their case, as well as in that of Ogden (1948), there was evidence of eclampsia. Shallow, Herbut, and Wagner (1946) considered that rupture of an arteriosclerotic vessel was the usual cause of abdominal apoplexy, but Brewer and Marcus (1948) were of the opinion that hæmorrhage is more probably due to a congenital aneurysmal defect in one of the vessels. Sherlock and Learmonth (1942) stated that in 14 cases of aneurysmal rupture occurring in pregnancy none showed toxæmia.

Danforth (1945), in reporting a fatal case of retroperitoneal hæmorrhage due to rupture of a splenic aneurysm, remarked on its extreme rarity and stated that only 13 cases had been recorded in the literature. Ogden (1948) could list only 6 cases of retroperitoneal hæmorrhage complicating pregnancy, but it appears that he must have overlooked a considerable number. Lennie and Sheehan (1942) collected in Glasgow 4 cases of ruptured aneurysm occurring during pregnancy during the course of ten years. Two of these affected the splenic artery and two the renal artery, but this relatively high incidence suggests that the diagnosis may often be missed.

Abdominal aneurysms, although rare, are frequently multiple and often occur at the point of bifurcation of a medium-sized artery. Several arteries may be affected. Machemer and Fuge (1939) were able to collect 84 cases of aneurysm of the splenic artery. Many of these had been diagnosed only at post-mortem examination, and Baumgartner and Thomas (1924) noted that 33 of the 40 splenic aneurysms collected in their series had only been recognized at autopsy. Schroeder (1918) found 20 cases of splenic aneurysm in 32,768 autopsies and Lindboe (1932) was able to demonstrate 26 further cases in 41,437 autopsies. Schuster (1937) noted that familial hæmorrhagic telangiectasia might be associated with multiple aneurysms of the splenic artery and quoted Greenfield, who believed that in relation to the cerebral vessels at least a hereditary tendency to aneurysm might be traced. Anderson and Gray (1929) believed that arteriosclerosis was responsible for some splenic aneurysms, but not for all, and Forbus (1930) stated that the cause of aneurysm was a failure of the media of the main arterial trunk to fuse with that of its branches which is formed later, with the result that aneurysm tended to appear at the junction of the main trunk and the branches. Shallow, Herbut, and Wagner (1946) found a defect of the internal elastic lamina and most of the media in their case in which multiple aneurysms were present in the inferior pancreatico-duodenal artery, and Schuster (1937) also demonstrated a characteristic loss of elastic tissue in the media of the splenic artery. She considered that an inborn vascular defect explained the development of aneurysms, and Low (1934) pointed out that in his case all the larger arteries were thin walled. Danforth (1945) found areas of necrosis in the intima and the adjacent media of the splenic artery in his patient. Sherlock and Learmonth (1942) found associated pathology present in 74 of the 125 cases of aneurysm which they collected; 46 per cent showed arteriosclerosis, and embolism, usually resulting from endocarditis, was found in 23 per cent. They considered that weakened areas at the bends in vessels might lead to bulging even in the absence of hypertension, but they quote Gunther, who found splenic aneurysm present in 4 out of 400 hypertensives examined post mortem. Sherlock and Learmonth (1942) also point out that aneurysm commonly occurs at the point where a main vessel undergoes branching or deviation.

Rupture of a splenic arterial aneurysm usually gives rise in the first place to retroperitoneal hæmorrhage. It may occur in two stages with primary formation of a false aneurysm whose walls consist of old blood-clot (Machemer and Fuge, 1939; Lennie and Sheehan, 1942; Gillam, 1942). This occurs in 12·5 per cent of cases (Sherlock and Learmonth, 1942). As it increases in size the posterior peritoneum is ruptured and the hæmatoma spreads to the lesser sac of peritoneum. If bleeding continues further blood may spill through the foramen of Winslow to the greater sac of peritoneum. Rupture of an aneurysm of the renal artery or hæmorrhage in the retroperitoneal space occurring from other causes may, of course, give rise to

bleeding of similar distribution. In a few cases hæmorrhage may remain more or less completely retroperitoneal, as in that reported by Kenny and Doniach (1945) and that of Low (1944), where only a little dark fluid was found in the peritoneal cavity. In the case of Potocki (1918) also there was no blood in the peritoneal cavity, but he remarks that he could find little reason why the hæmorrhage should remain retroperitoneal. Sherlock and Learmonth (1942) state that bleeding extends to the peritoneal cavity in 59 per cent of cases and that very large amounts may be present in this situation. Danforth (1945) found at post-mortem examination in his case not less than 3½ litres of blood in the abdominal cavity as well as a retroperitoneal hæmorrhage.

Hæmorrhage may be precipitated by trauma, and MacLeod and Maurice (1940) consider that some exertion is always present as a precipitating factor, although sometimes this may be indescribably slight. Hæmorrhage is more likely where hypertension is present, or where there has been eclampsia or pre-eclamptic toxæmia (Kenny and Doniach, 1945; Ogden, 1948). Coitus was the precipitating cause in the cases described by Potocki (1918) and Kenny and Doniach (1945). In the case of MacLeod and Maurice (1940) hæmorrhage followed a violent fit of laughter. Smith (1911) recorded a case where pain occurred after the patient had completed her weekly washing and mangling. In the cases of Gillam (1942) and Low (1944) rupture apparently followed bowel movement, and Eiss (1936) described constipation as a precipitating factor. The presence of a large intra-uterine tumour in the abdomen, especially in a primigravida with tense abdominal muscles, may lead to increased abdominal pressure and so to rupture. In Smith's case (1911) a vegetative endocarditis was present and he suggested that an embolus from the mitral valve may have become impacted in one of the smaller splenic vessels and caused either a local increase in pressure or a gradual weakening of the vessel wall. Sheehan and Falkiner (1948) observe that during the course of the pregnancy a number of patients have considerable splenic enlargement, usually associated with accidental hæmorrhage or with anæmia. They suggest that this enlargement is accompanied by some alteration of the blood-supply to the spleen and that this may be related to the high incidence of rupture of splenic aneurysm recorded in late pregnancy.

CLINICAL FEATURES

Before rupture occurs the presence of a splenic aneurysm may be suspected in cases of familial hæmorrhagic telangiectasia (Schuster, 1937), or where there is any family history of congenital aneurysm. The condition at this stage may be completely symptomless, but Sherlock and Learmonth (1942) have found such symptoms as vague dyspepsia, epigastric pain, vomiting, anorexia, and anæmia. These authors state that occasionally a tumour may be palpable which gives rise to a murmur audible on auscultation of the left hypochondrium.

Rupture may occur in two stages, as has previously been noted. A primary rupture into the retroperitoneal space, or even into the lesser sac of peritoneum, is not incompatible with temporary recovery, as in the case of Gillam (1942), but sooner or later a secondary rupture will prove fatal if operative interference is not undertaken. In the first stage the patient complains of upper abdominal pain with generalized tenderness and possibly epigastric fullness which may suggest an acute dilatation of the stomach (Gillam, 1942). At this stage auscultation of the upper abdomen may reveal the bruit of an aneurysm (Brockman, 1930), and this may determine laparotomy and splenectomy or ligation of the splenic artery. If this is not carried out a more complete rupture will take place sooner or later. Commonly the patient will complain of severe upper abdominal pain, which may follow exertion or trauma, and the onset of symptoms is rapidly followed by collapse, with characteristic signs of blood loss and shock, pallor, rapid thready pulse, sighing respiration (' air hunger '), etc. In certain cases the progress of the hæmorrhage is marked by the occurrence of repeated convulsive seizures, which in late pregnancy may be mistaken for eclampsia, especially with signs of abdominal hæmorrhage which may mimic concealed accidental hæmorrhage. The abdomen in the majority of cases where hæmorrhage occurs into the peritoneum becomes rigid and will give a dull note on percussion. There is extreme generalized tenderness, and in some cases a bulging may be noted in the left flank owing to the formation of a large retroperitoneal hæmatoma. Hæmatemesis and melæna may occur where rupture occurs into the stomach, as in the second case of Sherlock and Learmonth (1942).

Death may occur rapidly. In the case of Kenny and Doniach (1945) death ensued within two hours of the first symptoms, but the patient may survive for several days or even longer where the primary rupture merely gives rise to the formation of a false aneurysm. In the case of Gillam (1942) a primary rupture occurred apparently fourteen days before the secondary and caused in the first instance severe symptoms which, however, settled down. Laparotomy was successfully performed and splenectomy carried out after the secondary rupture. The uterus is not affected by the hæmorrhage, but it may give the appearance of a concealed accidental hæmorrhage because of the rigidity of the overlying abdominal muscles. The fœtal heart-sounds are usually absent.

The parity of the patient seems to be of little significance since cases have been reported in multiparæ as often as in primigravidæ.

DIAGNOSIS

The condition is rarely encountered until gross rupture has occurred, but it may be possible to identify the condition in the early stages by the history of dyspeptic symptoms and the presence of a tumour, as described by Sherlock and Learmonth (1942). Hogler (1920) demonstrated radiologically a pulsating shadow giving rise to a filling defect in the stomach, and Lindboe (1932) diagnosed a splenic aneurysm on demonstrating by radiography a sharply defined calcareous ring behind the stomach. Following rupture diagnosis may be very difficult, particularly during pregnancy where the site of the hæmorrhage is masked by the presence of a large uterus.

The signs of severe intra-abdominal hæmorrhage, however, should demand immediate laparotomy, and Lennie and Sheehan (1942) have stressed that even if only a probable diagnosis can be made evacuation of the clot and ligation of the bleeding vessels should be undertaken. The condition must be differentiated from spontaneous rupture of the uterus, concealed accidental hæmorrhage, and ectopic pregnancy. Coronary thrombosis and mesenteric infarction should be considered as well as perforation of a viscus, acute pancreatitis, and other acute abdominal catastrophes. Sperling (1940) states that only 3 cases have been diagnosed before operation or death, but Sherlock and Learmonth (1942) state that the diagnosis was made in 8·1 per cent of cases.

TREATMENT

Sherlock and Learmonth (1942) stress the importance of diagnosis before rupture since only then is surgery likely to be successful, but Gillam (1942) considers that in cases of two-stage rupture splenectomy gives an excellent chance of curing the condition if the diagnosis is made. Sherlock and Learmonth (1942) point out that the longest recorded interval between first symptoms and rupture is 14 years, and that this may give ample opportunity for diagnosis, but as diagnosis is always so difficult, in the majority of cases treatment will be usually called for after rupture has already occurred. Machemer and Fuge (1939) consider that at this stage the condition is so dangerous that only early diagnosis and proper treatment can prevent a tragic end and that treatment is obviously entirely surgical. Danforth (1945) states that in spite of the grave nature of the emergency immediate recognition and efficient surgical atttack may rescue the patient. In the presence of a pregnancy of more than four months' duration, the uterus must be emptied first to facilitate the approach to the bleeding vessel and the fœtus need not be considered as it is almost invariably dead when the patient is first seen. When the posterior abdomen is exposed rupture of a hepatic or mesenteric aneurysm should be considered, and then the splenic and renal vessels should be approached via the gastro-colic omentum. Digital compression of the splenic or renal artery may help to control bleeding while clot is evacuated and the bleeding point identified. If the diagnosis of splenic aneurysm is confirmed, the splenic artery should be ligated. The spleen may be removed or may be left to undergo aseptic atrophy (Lennie and Sheehan, 1942). Immediate operation is always called for in a case of this kind, but resuscitative measures, especially blood transfusion, should be applied at the earliest possible moment.

As an alternative to ligation, tamponade has been advocated (Latzko and Schiffmann, 1928), but this has never been successful and carries a mortality of 100 per cent (Danforth, 1945).

PROGNOSIS

Rupture of a splenic aneurysm is a grave abdominal catastrophe and only purposeful surgery can save the patient. The outlook is especially serious during pregnancy because of the difficulty of diagnosis and because of the presence in the abdomen of a large pregnant uterus which interferes with access to the bleeding vessel. Death may occur with startling rapidity, and MacLeod and Maurice (1940) note that the interval between the first symptom and the death of the patient in three recorded cases was 10, 15, and 90 minutes respectively. Only two cases are recorded as having survived after rupture of a splenic aneurysm associated with a pregnancy—namely, those of MacLeod and Maurice (1940) and of Gillam (1942). O'Connor and Bradley (1946) have also described a case where retroperitoneal hæmorrhage was associated with a pregnancy and the patient survived without operative interference other than Cæsarean section, but here the hæmorrhage was probably due to rupture of a hæmorrhoidal vessel and was restricted to the pelvis, the peritoneum apparently remaining intact. In the absence of pregnancy the outlook is better, and Lower and Farrell (1931) described 15 cases operated on with 7 recoveries. Berk, Rothschild, and Doane (1941) collected 20 cases of intra-abdominal apoplexy of various types where ligation was carried out in 80 per cent and recovery occurred in 69 per cent. Sherlock and Learmonth (1942) described 38 cases operated upon with 20 survivals, but in only 2 of these had prior rupture of an aneurysm taken place. Parsons (1937) ligated the splenic artery successfully in his case, but the patient died from rupture of a second aneurysm twenty-two months later. Danforth (1945) states that in pregnancy the mortality in cases where ligation is carried out should be about 25 per cent, but that owing to difficulties of diagnosis and of treatment this may not always be possible. Gillam (1948), reporting 2 cases of spontaneous rupture of the splenic artery, the first previously reported in 1942, observes that the second case probably owed his life to the experience gained from the first.

CASE REPORT

Mrs. M. U., aged 26 years, had had a normal pregnancy fourteen months before I saw her. Her previous health had always been good, but she had suffered from pain in the left hypochondrium at intervals for many years. This had never been severe and had never been fully investigated. She was in the thirty-ninth week of her second pregnancy when she was awakened from sleep about 3 a.m. with severe abdominal pain. She had until then been perfectly well, but the pain was so severe that she was forced to kneel up in bed until she collapsed. Several further attacks occurred during the night, but help was not summoned until about 8 a.m. when the District Nurse was called. In view of the repeated convulsive seizures the nurse considered that the condition was due to eclampsia and called medical aid. The patient's family doctor recognized that he was dealing with an acute abdominal emergency, but owing to the rigidity of the abdomen he was unable to make a diagnosis. The patient had been seen regularly for ante-natal examination and had shown no sign of pre-eclamptic toxæmia nor any other deviation from the normal.

I was called to see the patient about two hours later, but by the time I reached her, 70 miles away, about 12.20 p.m. she was exsanguinated and obviously moribund. She died before it was possible to start blood transfusion. The abdomen was by now relaxed owing to the exhaustion of the patient. Although the uterus was clearly outlined, the upper abdomen was somewhat full and doughy. There was no external hæmorrhage, but death was obviously due to internal hæmorrhage. Owing to the fact that Ogden's paper had been published a week or two before and that the letter of Brewer and Marcus

had appeared only a few days previously, a tentative diagnosis was made of rupture of a splenic arterial aneurysm as the fundamental cause of death and this was confirmed at post-mortem examination a few hours later.

At autopsy about 6 pints of rather watery blood were removed from the general peritoneal cavity. The lesser sac was filled with blood-clot to the amount of about

FIG. 130.—Spleen and pancreas seen from behind. Splenic vessels incised and aneurysm at bifurcation of splenic artery laid open. Rupture occurred at the point most remote from the artery. Note hæmorrhagic infiltration in the region of the tail of the pancreas.

1 pint, and when this was emptied a massive retroperitoneal hæmorrhage was found originating in the region of the hilum of the spleen and the body of the pancreas. The pregnant uterus was normal and the fœtus of about 39 weeks' maturity. There was no abnormality of the liver, but the retroperitoneal hæmatoma had infiltrated extensively in the region of the pancreas, the hilum of the spleen, and the retrogastric space. There appeared to be abnormality of the aorta or of the stomach. The spleen and pancreas, together with much of the infiltrated tissue in the area, were removed for further examination, and, with the aid of Dr. H. J. R. Kirkpatrick, a large aneurysm of the splenic artery was demonstrated near the point of bifurcation of the artery as it entered the hilum of the spleen. This was ovoid in shape, about 3 cm. in its long axis, 2 cm. broad, and 1 cm. in depth. It extended upwards from the upper surface of the artery and the point of rupture was clearly seen at the point most remote from the main artery (*Fig.* 130). The weight of the spleen was approximately 250 g. and so the organ was moderately enlarged.

DISCUSSION

Rupture of a splenic arterial aneurysm is fortunately rare, since it can be so rapidly fatal, especially where diagnosis and treatment are hampered by the presence in the abdomen of a large pregnant uterus. The case reported above would appear to be the fifteenth to be recorded and it is felt that it is important that every case of this type should be put on record in order that obstetricians and others may be reminded of this clinical entity. This is of enormous importance since the outcome depends very largely upon the promptitude with which diagnosis is made and treatment undertaken.

In the present case the patient had always appeared healthy, but a history was elicited of slight dyspepsia and epigastric pain, which may well have been due to the presence of a splenic aneurysm. No full investigation of this symptom had been undertaken and the condition remained undiagnosed until after rupture had occurred. In this instance nine hours elapsed between the onset of the first symptom and the death of the patient. It would appear that the formation of a false aneurysm did not occur here and that the hæmorrhage was rapidly progressive from the moment of rupture. In this type of case the prognosis is correspondingly worse than in those in which two-stage rupture occurs, where more time is given for diagnosis and treatment while the patient still has a reasonable chance of survival. Even in this case the delay of five hours before any help was summoned was probably fatal, since the coincidence of my recent study of the relevant literature had enabled me to reach the correct diagnosis, and, as Latzko and Schiffman (1928) point out, this is of major prognostic significance. Lennie and Sheehan (1942) have stressed that where even a probable diagnosis is made, drastic and heroic surgery is indicated, and had this patient been seen earlier it might have been possible to save her life as she lived at a distance of only one mile from a well-equipped local hospital. There was no suggestion in the history of any traumatic or other factor precipitating rupture of the aneurysm, since the patient had spent the evening sitting quietly at home, had gone to bed in the usual way, and had been awakened from sleep by her first symptoms.

The most probable explanation of rupture in this case is the gradual stretching of the aneurysm by the arterial blood-pressure, which may have been raised locally here as a result of the increased intra-abdominal tension due to the large size of the pregnant uterus nearing full term. The position of the rupture at the point farthest away from the artery suggests that a *locus minoris resistentiæ* developed in that area and had given way simply as a result of progressive over-stretching. No histological

studies of the aneurysm were undertaken since it was felt that defects of the media and of the elastic tissue of the vessel wall are characteristically found in almost all aneurysms.

The increased size of the spleen confirms the observation of Sheehan and Falkiner (1948) that in late pregnancy the spleen may be considerably enlarged, but in this case there was no accidental hæmorrhage and nothing to suggest an anæmia before the rupture of the aneurysm occurred. It seems therefore that this case can add little to indicate whether this splenomegaly of late pregnancy is a causal factor in splenic aneurysmal rupture, as these authors suggest.

A survey of the literature on the subject of splenic aneurysm in association with pregnancy reveals that the outlook for survival of the patient has been almost uniformly unfavourable and this has been due in most instances to failure to make the diagnosis of the condition. Even in Gillam's successfully treated case the treatment was undertaken only when a second rupture had occurred, the nature of the first rupture not having been recognized. It would appear that the only way in which the prognosis can be improved is by the fostering of a greater awareness of the condition amongst all practitioners and by a readiness to undertake extensive surgical treatment as soon as the diagnosis suggests itself as a possibility. Only then will it be possible to approach the mortality figure of 25 per cent quoted by Danforth as being the lowest attainable.

SUMMARY

1. Rupture of a splenic arterial aneurysm is one of several conditions which have been associated with severe intra-abdominal hæmorrhage during pregnancy.

2. Fourteen cases have been described in the literature and an additional case is reported.

3. Prognosis is bad, primarily because of diagnostic failure, but treatment also presents considerable technical difficulty.

4. The only factor likely to improve the prognosis is a greater awareness of the existence of the condition and consequent earlier diagnosis and adequate treatment.

I am indebted to Dr. H. J. R. Kirkpatrick for his assistance in the demonstration of the aneurysm; and to Mr. James Hall for his indefatigable efforts to obtain a satisfactory photograph of the specimen.

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A NEW URETERO-ENTEROSTOMY

BY G. C. THOMSON, JOHANNESBURG

THE operation of uretero-enterostomy is essentially associated with the name of Robert C. Coffey, and many variations of his original three techniques have been tried. The two-stage method is probably the most popular, but, although the risk is less, the strain on the patient is considerable. Transplantation of both ureters at one sitting is quite feasible but may lead to a stormy convalescence, and in the debilitated to a good deal of anxiety, before function is fully established. In the method submitted, both ureters are transplanted intact in one stage and the fistula from ureter to bowel is subsequently established by a minor procedure, without anæsthesia. The time interval between first and second stages varies and is left to the discretion of the surgeon.

The procedure is briefly as follows: After a preliminary intravenous pyelogram, sulphasuxidine therapy is instituted and the patient admitted a day or so prior to operation. If the bowels are regular, they are not interfered with, but an enema is given the night before where constipation is a factor. Premedication with omnopon and scopolamine is

used and the transplantation done under spinal anæsthesia.

On the table the patient is placed in a partial lithotomy position and then tilted into rather full Trendelenburg. With a subumbilical incision, the ureters are exposed in the usual way and the pelvic colon arranged conveniently between them. The anterior longitudinal muscle is split down to the mucosa, a matter of about 7–8 cm. Sutures are then inserted to fix the ureters without tension at the upper and lower extremities of the wound respectively. At this stage a sigmoidoscope is passed by an assistant and guided into position at the upper end of the incision in the bowel. A piece of 18-gauge copper wire, some 20 in. in length, is bent on itself in the form of a hairpin and the limbs pushed through the mucosa into the sigmoidoscope on either side of the ureter. With the loop controlled above, the assistant then twists the wires

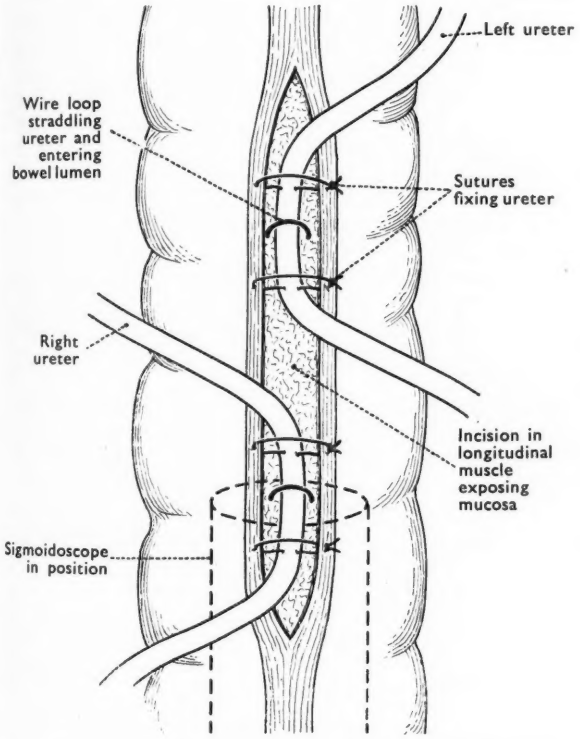
Left ureter
Wire loop straddling ureter and entering bowel lumen
Sutures fixing ureter
Right ureter
Incision in longitudinal muscle exposing mucosa
Sigmoidoscope in position

FIG. 131.—Method of fixing ureters in the submucosa. Anterior view.

until they are entwined up to the mucosa. A length of plastic tubing (P.V.C. sleeving) about 4 mm. in diameter is next pushed over the twisted wires, up to the point of their emergence from the mucosa into the lumen of the bowel, thus insulating them and preventing burns of the mucosa at the second stage, and the end is tied with silk securely to the wire. The other ureter is treated similarly before withdrawing the sigmoidoscope. One suture is inserted in the muscularis to steady the wire loop and avoid direct pressure on the ureter, which is then buried and peritonized in the usual way.

It is advisable to have the wire loops prepared beforehand and to sharpen the ends, and it is also wise for the assistant to be prepared to flush the bowel at the end of the sigmoidoscope, as good vision is essential to adjust the plastic tubes.

On the seventh day the diathermy machine is brought to the patient's bedside, the plate is fixed on the thigh, and the cutting current attached to the wire over either the left or right ureter. Gentle

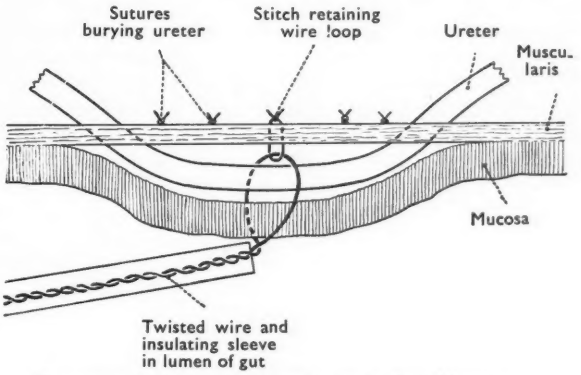

FIG. 132.—Relation of insulated wire in sigmoidoscope to ureter in submucosa. Lateral view.

traction on the wire while the current is flowing severs the ureter and underlying mucosa and establishes the fistula into the bowel. The cutting produces a feeling of heat and some abdominal discomfort afterwards, but does not hinder the patient getting up the same day. When function is established the second ureter is divided.

I do not propose to describe in detail the 6 cases. One case in the series died, but the error was essentially one of judgement in selection and not a fault in the method. The patient, an elderly African native, had an advanced carcinoma of the prostate with obstructive symptoms. Resection was the obvious choice, but in an effort to avoid local interference in the presence of malignancy ureteroenterostomy was done. In spite of free fluid being present in the abdomen the patient stood the operation well but subsequently developed pneumonia and died on the sixth post-operative day.

The original second-stage procedure consisted of introducing a sigmoidoscope in order to insulate the wires, but this proved extremely difficult in one case which had had pelvic peritonitis and three previous abdominal operations, and in which the wires had become crossed during twisting. The method was consequently abandoned in favour of the one described, in which the insulation is done under vision at the first stage.

The fifth case with a bladder malignancy involving the trigone and posterior urethra has undergone cystoprostatectomy subsequent to uretero-enterostomy and is progressing satisfactorily five months after the original operation.

An interesting feature of the most recent case in the series was that the patient passed urine per rectum within an hour of cutting the ureter.

SUMMARY

A new uretero-enterostomy is presented which claims to be relatively safe and entails only one major procedure.

DAVID FLEMING AND THE OPERATION FOR LIGATION OF THE CAROTID ARTERY

By J. J. KEEVIL, D.S.O., Surgeon Commander, R.N. (Retired)
KEEPER OF THE LIBRARY, ROYAL COLLEGE OF PHYSICIANS

David Fleming was very young, inexperienced, and obscure when in 1803 he was suddenly required to perform an operation of which it is improbable he had any previous knowledge, or of which only the most discouraging reports could have reached him, reports, moreover, carrying the high authority of John Abernethy.

Several factors caused his achievement to be overlooked both by his contemporaries and by posterity: Fleming was far from home at the time and, instead of the publicity that would have accompanied such an operation performed in a metropolitan hospital, it was done in the presence of a few seamen on the dimly lit deck of a great ship off the enemy coast. He may have intended to publish his experience later, but at the time he had no ready access to the medical press; his notes are unusually complete for an emergency operation at that period, but he may well have hesitated to publish a single case where the situation had forced him to employ a controversial technique; the nature of the operation was such that it is unlikely he had an opportunity to repeat it and thus to collect a series of cases suitable for an article in one of the gazettes or journals of his day. Fleming's early death supplies a further reason for the lack of recognition he received. The oversights of the past matter little where records are carefully preserved, but the Admiralty medical journals have in this, as in many other cases, now been destroyed, and we have only the evidence of his contemporaries to support a claim for Fleming that he was a pioneer in the ligature of the carotid artery.

David Fleming appears in naval records as acting Surgeon's Mate in the *Nymph* on April 27, 1793, his acting status resulting from his having been entered without an examination. It was not till July 4, 1793, that he appeared before the Court of Examiners at the Theatre of the Company of Surgeons and was passed Second Mate of a Third Rate. On the following day he was confirmed as Surgeon's Mate of the *Nymph* and remained in her till December, when he was transferred. He now joined the frigate, *Arethusa*, as Surgeon's Mate, and then after a year in her was moved to the frigate *Indefatigable*. This last change was probably a hurried one, for on Feb. 2, 1795, the day on which Fleming left the *Arethusa*, Captain Sir Edward Pellew was despatched in her from Falmouth for Brest to ascertain what had occurred to the French fleet there under Vice-Admiral Villaret-Joyeuse. Fleming remained in the *Indefatigable* until September, 1795, when he was appointed Surgeon of the *Chapman*. So far he had been in no engagements with the enemy, but in his next ship, *Seahorse*, which he joined at the close of 1796, he was to see more active service.

The frigate was shortly to form part of the force under Sir John Jervis which was blockading Cadiz and was to be based on Gibraltar, but in the spring of 1797 she was nearly lost when she went ashore off Europa Point; she required extensive repairs. During this period in harbour Fleming was able to send his more chronic cases to hospital, among them the Surgeon's Mate, Sellas Wells. He was only 23 years old and in very poor health, but Fleming must have regretted his loss for he had much work on board. His journal for this period is the only one that has survived; it shows that he recorded his cases with great care and that he devoted much time and thought to their treatment. There are several references to drunkenness, especially in connexion with injuries ashore; on the night of June 11 the most serious of these occurred—a case of stab-wound in the chest; but the objective accounts reflect little of the brutality of the times, the harshness of discipline, or the distant rumbles of the mutinies at home. One of his patients had "ulcers", but he had come from another ship and it is unlikely that the *Seahorse* had scurvy on board, for when Fleming joined her she was in home waters and had then cruised in the Channel; fresh provisions should have been available; moreover the issue of lemon juice had been authorized two years previously. While Fleming was making these entries in May, 1797, a new commanding officer had been appointed to the *Seahorse*, and with him Captain (afterwards Admiral Sir Thomas Francis) Fremantle brought his newly married wife, the former Betsey Wynne. St. Vincent had promised the honeymooning couple a ship that was due to return to England, but precedence was given to an invalid, Captain Oakes, and the Fremantles reluctantly installed themselves in the cabin of the *Seahorse*. Neither of them had liked the ship and it can hardly have been a happy ship's company that sailed from Gibraltar with Nelson on board on July 1, 1797. On the 3rd she lay off Cadiz while the port was bombarded from boats led by Nelson and Fremantle, but the attack failed; Betsey wrote in her diary, "I was anxious for Fremantle and did not go to bed until he returned"; probably she added somewhat to the anxieties of the surgeon, too, but fortunately Fremantle returned safely, only slightly wounded from a blow. On the 5th there was a further unsuccessful attack, and it must have been a weary and disappointed party that Fleming saw on their return at 4 a.m.; Lieutenant John Hornsey (*Hersey*) had nearly been killed, but Fleming records no casualties in his journal. Although it did not close officially till Aug. 17, 1797, the last entry is dated July 4. The fragmentary condition of the manuscript suggests that the last part may have become detached and so lost, but it is more likely that Fleming had no time to complete his record during the busy and discouraging days which followed this entry. On July 15 the squadron sailed for Teneriffe; St. Vincent, hearing that a rich galleon from Mexico was at anchor there, had ordered Nelson to proceed to the island, but on their arrival at Santa Cruz on

the 21st no treasure ship was seen. Mrs. Fremantle mistook her morning sickness for sea-sickness and was fretful on passage, and the day after their arrival she was once more to experience the strain of action when an unsuccessful attack was made on the town. While she waited for news she sought the company of the sailmaker's wife. On the night of July 24–25 Nelson led a further attack, but at 2 a.m. as they were landing, both he and Fremantle were wounded in the arm. The attack failed, but the terms secured by Captain Troubridge were honourable and included the care of the wounded in the hospital at Santa Cruz. Casualties had been heavy for so small a force, 44 being killed, 97 drowned, and 1 reported missing; 105 officers and men were wounded. Nelson did not return to the *Seahorse*, although she was the nearest ship, and the hæmorrhage from his wound was only controlled by the neckcloth which young Nisbet, his stepson, had tied round the arm. Fleming would have had to carry out the amputation had not Nelson's reluctance to return to the ship without news of Fremantle made him order the boat to pull on to *Theseus*. It thus fell to Thomas Eshelby* of that ship to perform the historic operation which he recorded so tersely in his journal.† Meanwhile Fremantle had been taken to H.M.S. *Zealous*, where he received first aid. At 4 a.m. he returned to his own ship and his anxious wife. Fleming found that two musket balls had passed through the right biceps muscle, but there was no injury to the bone and he decided on conservative treatment. Tuesday, July 25, was a busy day for him with fifteen wounded men to look after, two of whom were dying, and another officer, Lieutenant John Douglas, shot through the hand. With four men killed and so many wounded the frigate must have been short of hands and he can have had little help. Mrs. Fremantle records in her diary that she refused to visit the cockpit, though twice asked to do so by Fleming. Probably he received help from the sailmaker's wife and any other women who may have been on board. As late as 1816 the surgeon of H.M.S. *Glasgow*, William Stenhouse, reported how he had been thus assisted after the action off Algiers under Lord Exmouth.

It must be remembered that since her marriage in Naples in January, 1797, Mrs. Fremantle had been almost constantly at sea and repeatedly in action, that she was pregnant, and that her husband had received a wound which at that date often proved mortal. She may well have felt under a strain and been critical of treatment when Fleming came each day at noon to dress his commanding officer. On Aug. 4, Captain Troubridge came on board and "was quite angry to find Fremantle so low and weak. The Surgeon of the *Zealous* came on board they all agree to say he does not live high enough and abuse Fleming for not giving him Bark and Port wine, he took some in the course of the day and found himself much better"; but four days later she added "Bark does not agree with Fremantle". During this period, though Fremantle was in his cot, the ship was at sea and in action and his wife wrote: "The motion hurts his arm much, and the noise of the guns annoyed us both beyond conception". It was now decided to evacuate the wounded, including Nelson, to England in the *Seahorse*, but Fleming was not to remain in her for her journey home. The Admiral had decided to take Thomas Eshelby with him, and Betsey wrote in her diary, "I am glad of it as Fremantle has no great confidence in Fleming". Certainly his conservative treatment had caused his patient continuous pain, controlled only by laudanum, whereas Nelson was making light of the loss of his arm and sending cheerful notes to Mrs. Fremantle. Fremantle's long convalescence has made the editor of Betsey's diaries speculate on whether he would not have been better off with an amputation; even though the correctness of Fleming's treatment was more than confirmed when Fremantle landed at Portsmouth on Sept. 2. Mr. Fitzmaurice, of Haslar Hospital, and Mr. Leeds, of the *Brunswick*, came immediately to the Fremantles' lodging and, examining the wound, "found nothing alarming in the appearance of it". In fact, the last dressing was applied a month later. Fleming can have got little credit for all this, however, and had long since been forgotten. Possibly he was glad to leave the *Seahorse*, but when he was transferred to the *Emerald* on Aug. 18, 1797, he was joining a notoriously undisciplined ship. He cannot have felt much pleasure in his new appointment, which began with the Boatswain being hanged on Aug. 19. He saw more active service in the *Emerald*, however, and remained in her for two years. After Nelson's recovery in April, 1798, the Admiral was ordered by St. Vincent to watch Toulon, and the *Emerald* formed part of his command. She became separated in a gale on May 21, and thus missed the battle of Aboukir Bay some ten weeks later, but joined the fleet blockading Alexandria, and on June 19 was in action with the squadron which captured a French force off Cape Sicié. It is not recorded whether Fleming had to treat any casualties during this period, and for the remainder of his time in her the *Emerald* was not in action. His next ship was the *Impétueux*, a ship of the line which had been involved in the mutiny at Spithead on April 15, 1797. Detached from the Channel Fleet, she had now been ordered to join Lord Keith at Minorca, and Fleming went on board when she put in to Tetuan for water on July 26, 1799. In her he experienced more of the shadowing and chasing which formed the daily routine of ships in the Mediterranean command, but only saw action of a minor nature, once in June, 1800, off Quiberon Bay, and again two months later in the Ferrol expedition. Only the destroyed journals could show whether he performed any operations during this period; all that the existing records of his service show is that

* Thomas Eshelby had only been in *Theseus* for a few weeks, having joined her on May 27, 1797. He was probably under 30 when he performed the operation on Rear-Admiral Nelson, for he had only qualified on April 17, 1791, as Third Mate of a Third Rate when he was examined at the Surgeon's Company. He passed as Surgeon to a Fourth rate on Sept. 4, 1794. Betsey Fremantle described him as "a sensible young man" when he joined *Seahorse* with Nelson on Aug. 20, 1797.

† "Compound fracture of the right arm by a musket ball passing through a little above the elbow. An artery divided the arm was immediately amputated and the following give him . . ."

throughout his brief naval career he never served in a naval hospital. It is, however, possible that he studied in a civilian hospital during a brief period of unemployment after he left the *Impétueux* on April 15, 1802. The peace of Amiens had been signed the previous month and Fleming was unemployed until the uneasy truce was almost at an end. War was once more declared in May, 1803, and by then Fleming had already been recalled; on March 10, 1803, he was appointed to the *Tonnant*. She had been one of the large prizes at the battle of the Nile in 1798 and had been immediately put into commission as the greatest two-decker in the British Navy, forming with her 80 guns a formidable addition to the fleet. Whether Fleming's appointment to her was a mark of special confidence, or whether he had met her commanding officer, Sir Edmund Pellew, in 1795, in the *Arethusa*, and had been asked for by him, are matters for speculation. She was fitting out in Hamoaze when he joined and did not commission till six days later. By the time war was once more declared on May 16, she had moved no farther than Cawsand, but almost immediately she was ordered to cruise off the Spanish coast and in August was in action with the *Duguay*, *Trouin*, and *Guerrière*. Whatever the surgeon's activities the silence remains unbroken, however, until Oct. 9, 1803, when he made an entry in a now lost journal which began: " . . . Mark Jackson, a servant on board His Majesty's Ship *Tonnant*, attempted to commit suicide ". The man had cut his throat and when Fleming reached him was bleeding freely; unable to move him, he ligatured the bleeding vessels on the spot, but by the time he had done so Jackson was pulseless. The wound, 4 in. long, had divided " the trachea between the thyroid and cricoid cartilages, the two superior thyroid arteries, the internal jugular vein, and grazed the outer and muscular coats of the carotid artery ". Fleming then brought the divided parts into contact, securing the patient's head well forward on the chest. Two hours later he moved the man to the sick berth. Convalescence was satisfactory till Oct. 17, when, during a proxysm of coughing, the carotid artery burst and " torrents of blood issued from the mouth and nose ". Fleming's attempts to secure the artery through the wound failed owing to the vessel's friable state and he therefore cut down on it below the wound. He intended to tie it, although he " had never heard of such an operation being performed " and the patient was now *in extremis*. Jackson survived this operation and made an uninterrupted recovery. By Oct. 22 he was able to swallow, feeding having till then been by " nutrient glysters ". On Oct. 24, Fleming removed the ligature by dissecting through healthy granulation tissue. The man was on the sick list till Dec. 20, 1803, and on the attending list till March, 1804, when he was sent to Plymouth Hospital for invaliding.

The preservation of this fragmentary information from Fleming's journal is due to a contemporary naval surgeon, R. W. Coley.* In 1816 Dr. Coley, on the retired list and practising in Cheltenham, read the newly published translation of Philibert Joseph Roux's *A Narrative of a Journey to London, in 1814; or, a Parallel of the English and French Surgery*, where Roux said: " It must be confessed that the ligature of the common carotid and external iliac arteries, are incontestably the two finest triumphs of modern surgery." Roux then mentioned several eminent surgeons who had performed these operations, but not unnaturally made no reference to Fleming. Coley therefore wrote to the *Medico-chirurgical Journal and Review* on Nov. 14, 1816, to do " justice to the memory of a man, now no more, and lately belonging to a class of the profession whose merits are not duly appreciated, considering their opportunities for general and extensive practice ". Dr. Coley continued: " By comparing dates, I have reason to believe, the honour of performing the first successful operation of tying the carotid artery belongs to the late Mr. David Fleming, a naval surgeon; and in proof of this assertion, I send you a concise statement of the case, drawn up from his Journal, by permission of Dr. Harness,† of the Transport Board. I can bear testimony as to the result of the operation, for being First-Assistant Surgeon of the *Tonnant*, I accompanied the man to Plymouth Hospital in March, 1804." This concise statement is headed " Case of Rupture of the Carotid Artery, and Wounds of Several of its Branches, successfully treated by tying the common Trunk of the Carotid itself ", and then follows the extract from Fleming's lost journal, a portion of which has been quoted. Dr. Coley states that the success of the operation was " much talked of by naval officers in general, belonging to the Channel Fleet ", but the results were not published, as Fleming sailed for the East Indies the year after he performed it and fell a victim to the climate shortly afterwards. James Johnson,‡ the Editor of the *Medico-chirurgical Journal and Review*, and himself a former naval surgeon, added a note to Dr. Coley's

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* Richard Warren Coley was examined by the Court of Examiners of the Royal College of Surgeons in London on Jan. 6, 1804, passed First Mate of a First Rate and paid his guinea fee. Three days later he was appointed an assistant surgeon in the Navy. He not only served with Fleming in *Tonnant*, but was in *Culloden* from June 8, 1804 to April 1, 1805 as his First Mate. He left her on promotion to Surgeon, April 2, 1805, and appears to have retired Dec. 21, 1811.

† John Harness, M.D., F.L.S., was born in 1754 and joined the Navy in 1776. He was appointed a Commissioner of the Sick and Wounded Board in 1800, in which office he pressed for improvement in the conditions of service of naval surgeons. In 1793, as Physician to the Fleet in the Mediterranean, he had immediately introduced the issue of lemon juice for the prevention of scurvy. This was ten months before the Sick and Wounded Board recommended a general issue, and many considered that in this matter he deserved much of the credit which Sir Gilbert Blane later claimed for himself. This controversy is described in a " Biographical Memoir of Dr. John Harness ", published in the *Naval Chronicle* (1816), **35**, 265–80, reprinted in *J. R. nav. med. Serv.*, 1937, **23**, No. 1, 2–14.

‡ James Johnson, M.D. (St. Andrews), L.R.C.P. (London), entered the Navy in May, 1798, and saw much active service. His considerable experience of tropical conditions led to his most important work, *The Influence of Tropical Climates on European Constitutions*, in 1812. In 1814 he entered general practice. He

report to the effect that in 1804 Mr. Abernethy published his case of ligature of the carotid artery and that he, the Editor, therefore considered the claim to priority should be shared with him, adding: "We believe that this interesting case presents the first and only instance where the carotid artery was taken up after a rupture or wound of the artery itself, and life preserved. . . . Naval surgery may justly be proud of this operation. *Palmam qui meruit ferat.*" This closing statement by Johnson conforms with the known facts and justifies Coley's claim for Fleming that he was the first *successfully* to perform the operation. Abernethy's case was not only unsuccessful but was reported in such a manner as to discourage any further attempt to ligature the carotid artery. He published it in his *Surgical Observations* in 1804, but gives no exact date for the operation, which he performed at St. Bartholomew's Hospital. There he ligatured the left common carotid artery of a man who had been gored in the neck by a cow. The patient died thirty hours later with various cerebral symptoms, which can to-day be attributed to other causes, but which Abernethy interpreted as the result of the operation. He wrote: "Though the stopping of the supply of blood to the brain did not for several hours produce any apparent derangement in the functions of the organ, yet such a state was gradually occasioned by it, and which was attended like the effects of concussion of the brain, with inflammation." It was fortunate for Mark Jackson that Fleming operated on him before such a statement from such an authority appeared in print. If Samuel Cooper's statement as to the date of Abernethy's operation is correct, it was performed many years before it was reported, and it is fair to conclude that Abernethy had abandoned the method as a result of this single fatal case. Cooper, in his Hunterian Oration delivered in 1832, stated: "It was some time before the commencement of the present century that I saw him (Abernethy) tie the carotid artery, and it may be doubted whether the operation had ever been fairly done, at an earlier period." In spite of this claim for Abernethy the orator added a footnote to the published address in which he makes the conflicting remark: "It was Sir Astley Cooper who first took up the carotid artery for the cure of aneurism." It has not been possible to establish whether Sir Astley Cooper published his cases at the time or later, and in any case they are not relevant, since in them he was not dealing with "a rupture or wound of the artery itself". It, therefore, seems just to maintain the claim made for Fleming, whose case was clearly described, dated Oct. 9, 1803, and officially reported, even though the official report has not been preserved. Of his career subsequent to this date we know little beyond Dr. Coley's statement; the Surgeons' Register for this period at the Admiralty is more than usually uncommunicative and against his name has the single word "Dead", without so much as a date against the end of his service. The pay documents, however, reveal that he left the *Tonnant* on May 9, 1804, and was appointed a fortnight later to the *Culloden*. There follows a pencil note in the Surgeons' Servitude Record, "6 Sept., 1806", which may indicate the date of his death.

published a total of eleven books and was Editor of the *Medico-Chirurgical Journal*, later the *Medico-Chirurgical Journal and Review*. He was appointed Surgeon to H.R.H. the Duke of Clarence's Household and then Physician Extraordinary to the King on the Duke's accession. He died on Oct. 10, 1845, aged 69. His biography is in W. Munk's *Roll of the Royal College of Physicians*, 1878, **3**, 238–41.

I am indebted to J. L. Thornton, Esq., Librarian at the Medical College of St. Bartholomew's Hospital, for the references concerning Abernethy.

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SARCOMA OF THE URACHUS

REPORT OF A CASE AND BRIEF REVIEW OF THE SUBJECT

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THE subject of tumours of the urachus was reviewed by Begg in 1931. He collected 50 cases from the literature, and after rejecting 6 for various reasons, 44 remained for analysis. Of these 44 cases of urachal tumours, 6 were described as sarcomata. The writer has examined the reports of these 6 cases, and is of the opinion that 3 cannot be accepted. Two are attributed to Greig Smith (1897), who, in his book, *Abdominal Surgery*, mentioned that he had met with 2 cases of sarcoma of the urachus, but gave no further details. A third case, that of Villa (1902), was diagnosed by its naked-eye appearance, and no microscopical examination was made. To the 3 remaining cases should be added a fourth

which Begg appears to have overlooked. This is an interesting and well-described case reported by Fulle (1914). Since the publication of Begg's paper a number of reports of carcinoma of the urachus have appeared in the literature, but only one additional case of sarcoma has been presented—that of Ransom in 1933. This brings the total of cases of sarcoma of the urachus up to 5, and these are summarized below. The purpose of this paper is to present a sixth case.

1. In 1893 Frank reported a case of sarcoma which was thought to have its origin in the sheath of the urachus in a boy of 11 years. The boy complained of loss of appetite, loss of weight for several weeks, and two weeks before he was first seen a small mass had been noted at the umbilicus. Examination showed a hard, circumscribed thickening of the umbilical region. At operation, the recti and the vertex of the bladder were involved by direct extension, while metastatic nodules were found in the omentum. Histology showed the tumour to be a spindle-cell sarcoma.

2. Fulle's patient (1914) was a 4-year-old boy who was brought up to hospital by his parents after they had felt a tumour the size of a mandarine in the hypogastric region. Treatment was refused at first, but the parents returned with the child after a month, when the tumour had grown to three times its previous size. It extended to a point two finger-breadths below the umbilicus, was roughly ovoid in shape with a knobbly surface, of firm consistency, mobile in a lateral direction but not vertically, and was less evident when the abdominal muscles were in a state of contraction. There were no urinary symptoms. At operation a tumour the size of an ostrich egg was found projecting into the abdominal cavity from the anterior abdominal wall. The inferior pole was adherent to the bladder. The urachus and obliterated umbilical arteries were normal in their upper parts, but below they fused with the tumour. Many metastatic nodules were present in the omentum and parietal peritoneum, and one of these was removed for examination. The lesion was deemed inoperable and the abdomen closed. Two months after operation the abdomen was grossly distended. The tumour was much increased in size and there was a considerable amount of free fluid in the peritoneal cavity. Death took place three months after the laparotomy. Histological examination of the material removed showed the tumour was a round- and spindle-cell sarcoma.

3. Greig in 1927 reported the case of a woman of 30 who complained of a tumour in the suprapubic region for eight months. At operation the tumour lay in the extraperitoneal areolar tissue deep to the transversalis fascia. It was soft but solid, and was encapsulated by a thin membrane. It was easily enucleated and had no attachment to the umbilicus, but at its inferior extremity the delicate capsule concentrated to a fibrous strand which passed to the urinary bladder. This strand was divided. The patient was well and free from recurrence seven years later. Histologically the tumour was a small spindle-cell sarcoma.

4. Vöncken and Cambresier in 1927 reported the case of a woman, aged 30, who complained of a swelling below the umbilicus of two months' duration. On examination the tumour was found to be the size of a small orange and it was fixed and tender. At operation the tumour, which was adherent to the posterior rectus sheath, was removed without opening the peritoneal cavity. Histological examination showed it to be a fibrosarcoma.

5. Ransom's case (1933) is of special interest because of the age of the patient—a female of 4 months—and also because the tumour was found to be derived from striped muscle. The child was brought up because for one month it had been noticed that a palpable tumour below the umbilicus was increasing in size rapidly. On examination a roughly spherical mass was felt in the abdomen arising from the pelvis and extending to the costal margin. A diagnosis of cyst of the urachus was made and the abdomen explored. The abdominal cavity was almost completely occupied by a tumour measuring 11·5 cm. in length attached to the anterior abdominal wall from the umbilicus to the apex of the bladder. This attachment was divided, the tumour removed, and the peritoneum over the bladder reconstructed. A course of X-ray irradiation was given after the operation, and two years later the child was in excellent health with no evidence of recurrence. On pathological examination the tumour was firm and solid in consistency, provided with a definite capsule, and homogeneous on section. Histology showed that it was a spindle-cell sarcoma, the prevailing cell-type being an immature striped muscle cell, i.e., a spindle-cell rhabdomyosarcoma.

Criteria for Diagnosis of Sarcoma of the Urachus.—At birth the umbilical arteries meet the urachus at the umbilicus and form the apex of a triangle whose base is at the puboprostatic ligaments (*Fig.* 133). After birth the urachus descends and in the adult the apex of the triangle comes to lie at a point usually situated at the junction of the middle and lower thirds of a line joining the umbilicus to the bladder apex. The two obliterated umbilical arteries are drawn together above this to form a continuous band, the ligamentum commune, passing from the junction to the umbilicus. Begg points out that any tumour arising above the junction of the urachus and the obliterated umbilical arteries cannot strictly be described as a tumour of the urachus, but arises most probably from the tissues of the obliterated umbilical arteries. He also shows that the ten tumours of mesodermal origin in his series all developed some distance above the bladder, and argues therefore that these tumours did not, in fact, arise from the urachus at all, but probably took origin from the strands or ligaments connecting the urachus to the umbilicus. Begg went so far as to claim that " true tumours of the urachus always have an epithelial element, and that those which did not satisfy this criterion should be discarded from the category ". Whether this conclusion is completely valid or not is doubtful, but Begg is certainly correct in insisting that tumours arising in the region of the urachus should not be assumed to originate in that structure unless there is reasonable proof. In more than one of the recorded cases of sarcoma of the urachus the tumour was so large that it must have been difficult to be certain of its exact origin. The writer suggests that the following criteria should be

satisfied before a tumour is accepted as a sarcoma of the urachus :—

1. The tumour must occupy a site consistent with the anatomical position of the urachus at the age of the patient.

2. There should be a demonstrable anatomical relationship between the tumour and the urachus.

3. The tumour must possess the pathological and histological features of a sarcoma.

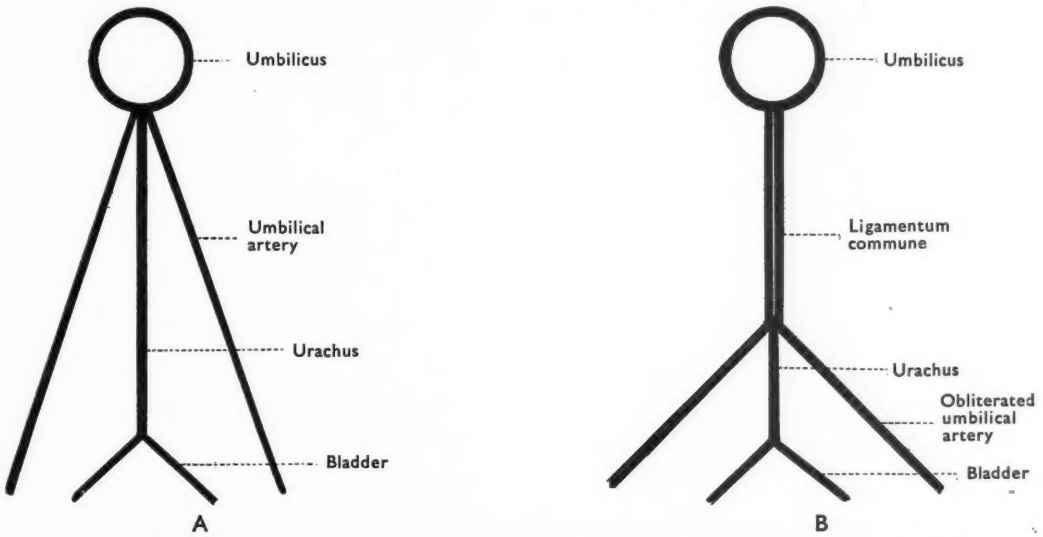

FIG. 133.—Relationship of the umbilical arteries to the urachus, (A) at birth and (B) in the adult. (*After Begg.*)

Re-examining the cases summarized above in the light of these criteria, it is seen that in no instance are they completely satisfied, so that it is doubtful if a completely substantiated case of sarcoma of the urachus has yet been reported. None the less, the evidence in the cases of Greig, Fulle, and Ransom, although not conclusive, is such that it is very likely that they were, in fact, examples of this condition.

CASE REPORT

HISTORY.—A boy of 6½ years was admitted to the West Middlesex County Hospital on Jan. 19, 1948, complaining of abdominal pain of three days' duration. At the onset the pain was in the umbilical region, but the next day it was felt in the right iliac fossa, and remained there until his admission. There had been some nausea but no vomiting. His bowels had been regular and micturition was normal. His previous health had been good.

PHYSICAL SIGNS.—The patient was an intelligent boy who appeared somewhat flushed. T. 100·4° F.; P. 120/min.; R. 22/min. Routine examination of the urine showed nothing abnormal. The tongue was clean. The respiratory excursion of the abdomen was diminished, and on palpation there was well-marked tenderness and muscle guarding localized to the right iliac fossa. On rectal examination palpation of the right side of the rectovesical pouch caused pain referred to the right iliac fossa.

A diagnosis of acute appendicitis was made.

OPERATION.—A few hours after admission the abdomen was opened through an oblique pararectal incision in the right iliac fossa. As soon as the peritoneum was incised some dark-red blood flowed into the wound. The appendix was a little injected. On passing a finger into the wound a tumour was felt attached to the lower part of the anterior abdominal wall in the midline, and projecting into the peritoneal cavity. After extending the incision medially the tumour was delivered into the wound. It was situated just above the apex of the bladder, but was separate from that organ. At the upper pole of the tumour was an ulcerated area covered with blood-clot, and this was oozing blood. The peritoneum was incised around the base of the tumour, which was then easily dissected off the apex of the bladder. In doing so a number of blood-vessels were divided and the urachus was severed twice—once where it entered the tumour below and again where it emerged higher up. The peritoneum was drawn together over the raw area. A finger was then passed into the upper abdomen and a mass was felt attached to the omentum. On delivery into the wound this was found to be some old blood-clot about the size of a grape and enclosed by a layer of fibrinous material. This had undoubtedly at one time been attached to the ulcerated area of the tumour. The portion of omentum containing the clot was removed and after appendicectomy the abdomen was closed.

PATHOLOGY.—The tumour (*Fig.* 134) measured approximately 5½ × 3½ cm. The lower part was spherical, firm in consistency, and was enclosed by a fairly thick white capsule. Antero-inferiorly this part was denuded of peritoneum, and here the urachus could be seen entering the tumour at its lower extremity and emerging higher up. The upper part of the tumour, which was somewhat larger, clearly consisted of tumour tissue which had erupted through the capsule. It had a greyish translucent appearance, was soft and friable, and its surface was smooth and undulating except over an area of ulceration where there was some adherent blood-clot. The cut surface of the tumour was greyish-white, semi-translucent, and homogeneous. Dr. A. C. Counsel reported on the histological appearances (*Fig.* 135) as follows: "There is some variation in structure in different parts of the tumour. The more cellular parts show fasciculi of spindle cells cut in various planes with darkly staining elongated nuclei. The nuclei are fairly uniform in size, but several mitotic figures are present in each high-power field. Vessels are seen as slits in the tissue lined by a definite layer of endothelial cells. The less cellular parts of the tumour show an œdematous increase in the stroma. There is no evidence of collagen formation. The tumour is a spindle-cell sarcoma. A section of the mass found attached to the omentum showed neoplastic tissue similar to that of the main tumour."

SUBSEQUENT PROGRESS.—Convalescence after operation was uneventful and the patient was discharged home ten days later. A radiograph of the chest revealed no evidence of pulmonary metastases. A course of deep X-ray treatment was subsequently given to the abdomen, and this was completed on April 21, 1948. The patient appeared to be in good health during the next few weeks, but on June 15, 1948, he was readmitted with a ten-day history of abdominal pain, swelling of the lower abdomen,

FIG. 134.—Naked-eye appearance of the tumour. At its lower extremity may be seen the cut end of the urachus. (× 1½.)

and pain in the right leg. There was some frequency of micturition. On admission his temperature was 100·6° F. and pulse-rate 128/min. He was miserable and in pain, and had lost some weight. A firm mass was palpable in the right side of the abdomen extending from the pelvis up to a level a little above the umbilicus. The mass was also palpable on rectal examination. From this point his condition deteriorated rapidly. The mass increased in size until it almost filled the abdomen and ascites made its appearance. Emaciation became marked and the temperature chart showed an irregular pyrexia. A second course of deep X-ray therapy to the abdomen was begun, but after two treatments the parents asked for the discharge of the child. He died at home on Aug. 10, 1948, seven months after the onset of symptoms. No post-mortem examination was made.

Comment.—In this case the criteria suggested above are satisfied. The tumour occupied the anatomical position of the urachus, and that structure was incorporated in the tumour substance. The histological characters and clinical course were those of a rapidly growing sarcoma. There seems no reason to doubt, therefore, that this is a true instance of sarcoma of the urachus.

The mode of production of the symptoms is of some interest. The onset of pain was due to bleeding into the peritoneal cavity resulting from the separation of omentum from the ulcerated area of the tumour. This separation may have been caused by some strain or exertion. In its mode of onset this case differs from the cases previously reported as sarcoma of the urachus, in which (with one exception) the presenting symptom was that of a palpable tumour in the hypogastrium or umbilical region. In other respects this case closely resembles that described by Fulle. In both instances the patient was a young child, the tumour was situated at, or just above,

FIG. 135.—Microphotograph of the tumour. (× 250.)

the apex of the bladder, and it was the seat of rapid growth and early massive intraperitoneal metastasis.

SUMMARY

1. The literature dealing with sarcoma of the urachus is reviewed.

2. Criteria for the establishment of a diagnosis of sarcoma of the urachus are suggested.

3. An additional case is reported.

I wish to thank Mr. John Scholefield, under whose care the patient was admitted, for advice and for permission to publish this case; and also Dr. A. C. Counsel for the histological report, Miss S. Robinson for the illustration, and Mr. D. A. Vinten for the photographs.

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PANCREATIC CYSTS

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PANCREATIC cysts are rare. At the Lahey Clinic there were only 16 cases from 1936 to 1948 (Warren, 1948). Although there is now an extensive literature on this subject, much of it is a record of individual experiences with small series of cases.

Jordan Lloyd (1892) first differentiated the pseudo-cysts of the pancreas from the true cysts. Korte (1898) defined the pseudo-cyst as a collection of fluid found in more or less close proximity to the pancreas, but not originating in the substance of the gland. This definition, widely quoted as it is, is inaccurate and has been responsible for some errors of classification by subsequent writers. Two examples of obvious inconsistencies of this definition will serve to illustrate its defects. The retroperitoneal cysts have many features in common with the mesenteric cysts, and they would not be classified as pseudo-pancreatic cysts even if they happened to be sited close to the pancreas. Yet such a cyst would satisfy the requirements of Korte's definition. On the other hand, an effusion of fluid into the substance of the pancreas is undoubtedly one variety of pseudo-pancreatic cyst, and yet Korte's definition would specifically exclude it from being so classified. The intent of Jordan Lloyd's classification is preserved if the pseudo-cyst is defined as a collection of fluid produced by the action of extravasated pancreatic ferments on tissues with which they come in contact. Such a collection of fluid could occur into the lesser sac of the peritoneum, it could occur into the peripancreatic cellular tissues, or even into the substance of the pancreas itself. A collection of blood or serum at these sites would not constitute a pseudo-pancreatic cyst. There must be production of fluid by the action of extravasated pancreatic ferments.

Jordan Lloyd recognized that trauma could produce a pseudo-cyst, and his first two cases give a clear history of trauma. Korte found a definite history of trauma in 33 out of 117 recorded cases. Lazarus (1904) recorded that he had produced a cyst containing 100 c.c. of milky fluid by crushing the pancreas of a dog. As milky fluid is rarely seen in pseudo-pancreatic cysts, it is more likely that this was an example of a retroperitoneal cyst produced by trauma to the lacteals.

Trauma is, however, not the only causative factor of pseudo-pancreatic cysts. Acute or subacute pancreatitis causes necrosis of the pancreas with extravasation of the ferments of the pancreas. The stage is set for the production of a pseudo-pancreatic cyst. The fluid content would vary with the acuteness of the necrosis, acute pancreatitis giving hæmorrhagic effusions whilst subacute pancreatitis would give clear and dark fluids which might contain necrotic debris. Suppuration would be rare, and if it did occur the collection of pus would be better classified as one of the types of subphrenic abscess. Mild recurrent bouts of subacute pancreatitis are not uncommon, and a pseudo-pancreatic cyst could form as a sequel of several such attacks. The history of repeated episodes of epigastric pain preceding the formation of the cyst would clearly indicate the mode of formation of such a cyst.

That chronic pancreatitis could give rise to a pseudo-cyst is not so clear. Fibrosis of the gland could constrict the ducts and give rise to retention cysts. If pancreatic ferments exuded through the walls of these retention cysts they could cause an effusion of fluid which would constitute a pseudo-cyst. There are, however, no convincing case records bearing out this theory.

The true cysts of the pancreas have an epithelial lining. Mayo Robson and Moynihan (1903) recognized three classes of true cysts, the retention cysts, the proliferation cysts, and the cysts of congenital cystic disease, and this classification has received general acceptance. The proliferation cysts are true tumours. They comprise the cystadenomas and the cystic epitheliomas. They are rare. Cumston (1903) was only able to collect records of 15 cases. These cysts are multilocular and conglomerate and are separable from the pancreas. They are very similar both histologically and microscopically to the multilocular cysts of the ovary. The cysts are lined by tall, columnar, mucoid cells, which are replaced by a few layers of spheroidal epithelium in places. Irregular masses of spheroidal cells may be seen in the interstitial tissues, making differentiation between a cystadenoma and a cystic epithelioma difficult. In many cases a cystic epithelioma is only recognized when it has recurred after operative enucleation or when metastatic deposits are noted when the lesion is exposed at operation. The proliferation cysts are recognized by their forming a conglomerate polycystic mass which is separable from the pancreas, thus contrasting with the retention cysts, which are embedded in the substance of the pancreas, and the cysts of congenital cystic disease, which are scattered throughout the gland and are firmly attached to the pancreas.

The retention cysts of the pancreas, as the name implies, are cysts produced by cystic dilation of the ducts of the pancreas proximal to an obstruction of the duct. Virchow (1863) described a rosary-like dilatation of the whole duct system, to which he gave the name 'Ranula pancreatica' and Klebs suggested the name 'Acne pancreatica' for a similar condition in which there were numerous small enlargements of the minute ducts of the pancreas. Virchow also described a retention cyst in which obstruction of a main duct gave rise to a single cyst reaching the size of a fist. McCann (1913) recorded a case of this kind the size of a football. Experimental ligature of a pancreatic duct will not give rise to a retention cyst. Such a ligature causes the parenchyma of the gland to atrophy, but the islets of Langerhans persist. However, gradual obstruction of a duct by fibrosis would not be comparable with sudden obstruction by a ligature,

nor would it be comparable with intermittent obstruction of a duct by calculi. Demonstration of such mechanisms of obstruction have not been possible at operation. A communication between the duct system of the gland and the lumen of the cyst has, however, been shown at operation by Ludolph (1890), Dixon (1884), and Hagenbach. Usually the recognition of a large cyst as a retention cyst is based on the fact that it is unilocular, and if there is more than one cyst, the cysts would be scattered in the substance of the gland and would not form a conglomerate mass as is the case with the cystadenomas and cystic epitheliomas. Walton (1923) considers that demonstration of pancreatic tissue in the wall of a cyst will distinguish the retention cyst from a pseudo-cyst, but this is surely a very good example of an error perpetuated by Korte's definition of a pseudo-cyst. The retention cyst has a single lining of tall columnar or secondary flattened epithelium. It is distinguishable from a pseudocyst by its epithelial lining and from the proliferation cyst by being embedded in the pancreas and being unilocular. In congenital cystic disease a single cyst might not be distinguishable from the retention cyst till cysts developed in other organs.

The congenital cysts of the pancreas are of great interest. Pathologists have proposed the name ' dysontogenetic cysts ' for this group. These cysts occur most commonly in the kidneys, and such concomitant kidney cysts would be present in nearly all cases of dysontogenetic cysts of the pancreas. Lindau (1926), in his classical monograph on cerebellar cysts and hæmangiomas of the retina, observed that dysontogenic cysts of the pancreas were present in more than half the cases. The converse of this proposition is, however, not likely to be true. The dysontogenetic cysts of the pancreas are usually multiple and are scattered throughout the pancreas, thus contrasting with the conglomerate masses of the proliferation cysts. The interstitial tissue is increased, thereby reducing the parenchyma. The islands of Langherhans are intact. The cysts are lined by cuboidal cells, which become tall and columnar if there are papillary projections into the lumen of the cyst.

TREATMENT

The evolution of the different methods of treatment of pancreatic cysts is an interesting chapter in surgery. The ideal treatment of a cyst is excision. Unfortunately excision has proved to be a dangerous ' cure all ' for pancreatic cysts. The separation of the cyst wall from adherent vascular omenta and mesenteries could lead to alarming and dangerous hæmorrhage which has sometimes proved fatal. Guleke and Goebell assess the mortality of excision of pancreatic cysts at 10·7 per cent, and in cases in which extirpation proved to be impracticable during the course of the operation, the mortality rises to the alarming figure of 55·5 per cent (Gross, Guleke, and Kuttner). Indeed the pseudo-pancreatic cysts cannot be excised. They are walled in by neighbouring fibrosed mesenteries and omenta, and any attempt at dissection of these fibrosed walls would be fraught with grave risk of injury to the blood-vessels in these structures. On the other hand, proliferation cysts should be excised wherever possible. These multilocular cysts could not be dealt with satisfactorily by any method other than excision. Moreover the cystadenomas might harbour areas of malignant degeneration, and the cystic epitheliomas will not recur if they are extirpated before dissemination has occured. Fortunately the proliferation cysts are readily separable from the neighbouring structures, and excision is a practicable method of treatment.

Those cysts which cannot be excised must perforce be treated by other measures. Jordon Lloyd introduced simple external drainage for the treatment of pseudo-cysts, a method which proved quite successful in his second case. Unfortunately simple external drainage sometimes resulted in recurrence of the cyst from too early healing. Gussenbauer, a pupil of Billroth, introduced the method of marsupialization in 1882 in order to ensure more prolonged drainage. The skin was sutured to the parietal and visceral peritoneal layers overlaying the cyst, the cyst wall and its overlaying visceral peritoneum was then sutured to the parietal peritoneum. The cyst was opened and drained within the area demarcated by these sutures. Pinkham (1945) reported 10 successive cases of pseudo-pancreatic cysts treated by either simple drainage or marsupialization. Unfortunately even marsupialization sometimes fails. An obstinate fistula may persist, resisting cure by either cauterization or excision, and the persistant discharge sometimes excoriates the skin and inflames the tissues in the neighbourhood of the fistulous track. Serious inanition and even death have resulted from excessive drain of body fluids from such fistulæ. Secondary infection of the cyst cavity may further complicate the situation. Rössing estimates the mortality of marsupialization at 4·5 per cent. Marsupialization is more likely to result in a fistula in the more chronic cysts with thick, fibrosed walls. Marsupialization of a true cyst should invariably lead to a permanent fistula.

The permanent fistulæ sometimes resulting from marsupialization suggested the idea of internal drainage by anastomosis of the fistula to an upper abdominal hollow viscus. The first operation of this kind was performed by Kehr, who anastomosed the fistula to the gall-bladder and then anastomosed the gall-bladder to the stomach. Other workers introduced anastomosis of the fistula to the stomach, duodenum, or jejunum. The next advance was the planning of a deliberate two-stage operation in which the fistula established at the first operation was anastomosed to some part of the upper alimentary tract at the second operation. The long and tedious course taken for these two-stage operations, suggested the alternative of a one-stage anastomosis of the cyst to the stomach, duodenum, jejunum, or gall-bladder. Rudolf Jedlicka (1922) published a report on a case operated on in 1915 in which a stalk or infundibulum of a pseudo-pancreatic cyst was anastomosed with the stomach, this internal drainage being supplemented by external drainage of the cyst. This operation, the first deliberate one-stage internal drainage of a pancreatic cyst, is a landmark in the history of treatment of pancreatic cysts. Most pancreatic cysts lack an infundibulum suitable

for utilization for an anastomosis, and the technical details of Jedlicka's operations are therefore not applicable to most cysts, but the success of his operation served to introduce the important measure of primary internal drainage in the treatment of these cysts. The method of internal drainage was put on a firm basis by Jurasz (1931), who, using the transgastric route, made a large opening into the cyst through the posterior gastric wall, which was firmly fused with the wall of the cyst. So firm was the fusion between the posterior gastric wall and the cyst, that no sutures were needed to maintain apposition of the cut edges. The patient made an uneventful recovery, the operation wounds healing by first intention. The Jurasz operation is applicable to most pancreatic cysts, as the posterior gastric wall is usually fused with the anterior cyst wall. No adhesions are separated, there is no risk of hæmorrhage from separation of the cyst wall from neighbouring structures, access is easy, and the operation is completed in a short time.

The advantage of the Jurasz operation is not appreciated by many workers. A quotation from a recent paper on pseudo-pancreatic cysts by Bickford (1948) illustrates this point very well. "Case II. It [the pseudo-cyst] filled the lesser peritoneal sac, and the stomach and the transverse colon were spread out over its anterior wall to which they were intimately adherent, and from which they could not be separated without causing troublesome bleeding. It did not appear to be possible to perform an anastomosis between the cyst and the stomach or jejunum." This patient was treated by external drainage and the fistula took seventy-nine days to heal. The conditions were ideal for the Jurasz operation, which would have relieved the patient of his cyst without the inconvenience of an external fistula. Kerschner (1929) introduced a similar technique for one-stage internal drainage of a pancreatic cyst through the posterior duodenal wall, utilizing the transduodenal route. The operation would be applicable to the occasional case where the duodenum was more intimately adherent to the cyst than the stomach. In both these operations, the anastomosis is wide, exceeding $2\frac{1}{2}$ in. in length. Sutures need only be put in the anastomotic line for hæmostatic purposes, and in most cases such sutures are not necessary. The theoretical possibility that with so wide an anastomosis, gastric or duodenal fluids would flow into the cyst space, there to produce digestion and irritation of the cyst wall, has not been observed in cases subjected to these operations. X-ray examination after a barium meal shows that there is no regurgitation of gastric or duodenal contents into the cyst space. External drainage of a pseudo-cyst often results in obliteration of the cyst space by fusion of the adjacent walls of the cyst. There is no reason to suppose that this could not occur with internal drainage of pseudo-cysts. The Jurasz and Kerschner operations have, however, proved to be equally successful in the treatment of large unilocular true cysts of the pancreas, and as fusion of the cyst walls could not be expected in these cases, the good results must be attributed to the cyst space remaining as a potential space whose walls are always in apposition.

To sum up, there is a choice of treatment in regard to the large unilocular pancreatic cysts. It is not always possible to determine whether a cyst is a true or a pseudo-cyst even when it has been exposed to view at an operation, and the decision whether a cyst should be excised or drained should be made after a cautious attempt has demonstrated the possibility or impracticability of excision in any particular case. In regard to drainage, external drainage has the disadvantage of producing a fistula, which always takes a fair time to heal, and might even never heal. Internal drainage by the technique devised by Jurasz and also utilized in the Kerschner operation is simple, safe, and efficient, and rids the patient of his cyst in a one-stage operation healing by first intention.

CASE REPORTS

Case 1.—

HISTORY.—R. S. M. A. P., aged 30, female married Sinhalese, was admitted to the General Hospital, Colombo, on Nov. 25, 1947, for abdominal pain of three days' duration. The pain was a dull ache; it was felt diffusely and deeply within the abdominal cavity. It became more intense five minutes after a meal, and persisted as a severe pain for about two hours. She had vomited once on the day of onset, and again on the following day. The patient had also noted a lump in the upper abdomen for the last four years. It had started as a small lump lying to the left of the middle line and had steadily increased in size since then. Appendix removed four years ago. One year's malarial fever thirteen years ago. Married eight years. No children, periods regular.

ON ADMISSION.—The patient had a temperature of 100° and a pulse of 90. The abdomen was soft and there was a large round tumour the size of a grape-fruit in the upper abdomen lying more to the left than to the right side. It was well defined and painless and could be moved from side to side. It lay immediately below the anterior abdominal wall, but it could be ballotted into the left loin. It moved on respiration. It was dull on percussion. There was no increase in the area of splenic dullness. The tumour appeared to be a cyst from its shape and consistence. Its position showed that it could not have arisen from either the liver or the spleen, nor could it have been a cyst arising from the pelvis. From its position it was considered that the cyst must be a pancreatic cyst, despite its movements on respiration.

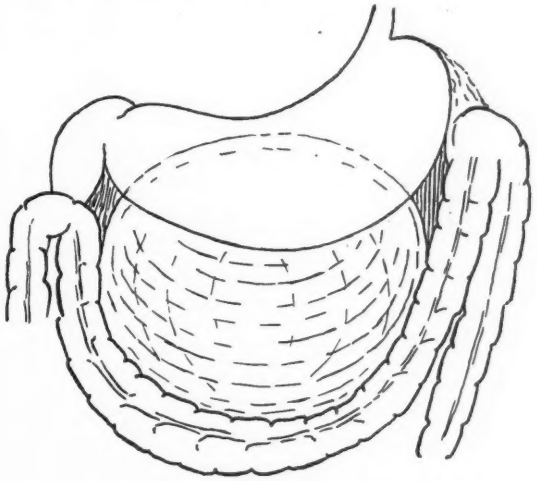

FIG. 136.—Diagram showing the relation of the cyst as seen at operation in *Case* 1.

Although it could be ballotted into the left loin, it lay so much nearer the anterior abdominal wall as to make its origin from the left kidney unlikely.

The connexion between the cyst and the attack of abdominal pain and fever was obscure. The pyrexia settled in three days and the patient was advised to have an operation for the cyst.

Nov. 27: *Blood-count*: R.B.C. 3,240,000; W.B.C. 10,000. Polymorphs 79 per cent, leucocytes 20 per cent, eosinophils 1 per cent.

Nov. 30: Urine, no albumin. No sugar.

AT OPERATION (Dec. 1).—Under light percaine spinal anæsthesia. Left upper abdominal paramedian incision extending to one inch below the umbilicus. Peritoneal cavity opened. Large spherical, thick white-walled cyst

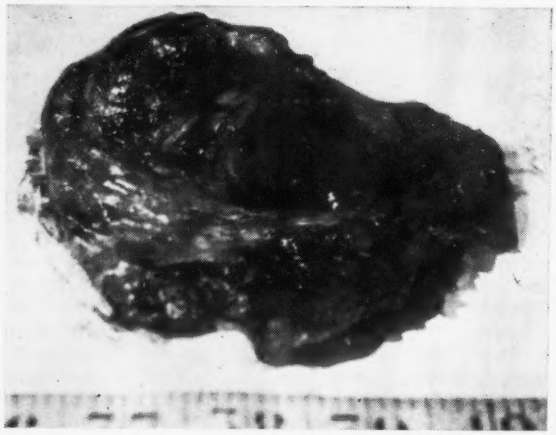

FIG. 137.—Photograph of the cyst removed in *Case* 1. The cyst has been artificially distended with gauze, but is much smaller than it was in situ.

was seen projecting forwards between the stomach and the transverse colon and covered by the gastrocolic omentum (*Figs.* 136, 137). The stomach, duodenum, gall-bladder, liver, and spleen, were all normal. The gastrocolic ligament was divided between ligatures and the cyst wall was now seen to be covered by the stretched peritoneum of the posterior abdominal wall. This peritoneum was incised and the cyst was gently enucleated from its bed. The cyst wall was greatly thinned at certain places, and the wall tore during enucleation. An opalescent mucoid fluid which had a definite sheen leaked from the cyst. At places the cyst wall was thick and there were multilocular cysts the size of marbles in the thickness of the cyst wall. Two blood-vessels passing posteriorly from the cyst bed to the cyst wall needed ligature. The inner cyst wall was flesh coloured, giving an appearance resembling the mucosa of a hollow viscus. The bed of the cyst was composed of compressed connective tissue, which had replaced the pancreas to such an extent that the aorta appeared not to be covered by the pancreas at this place. The head of the pancreas was felt to the right of the cyst bed within the curve of the duodenum. The incision in the peritoneum overlying the cyst was sutured and the abdominal wall was repaired without drainage.

The patient made an uninterrupted recovery from the operation, leaving hospital on Dec. 16, 1947.

HISTOLOGICAL EXAMINATION OF THE CYST WALL (W. A. E. Karunaratne).—The appearance is that of a cystadenoma lined by mucinous or pseudo-mucinous epithelium (*Fig.* 138).

The fluid from the cyst was sent for examination.

Reaction, alkaline. Urea, 0·2 g. per cent. Diastase, 100·0 units. No cholesterol crystals seen.

FOLLOW-UP NOTES.—The patient was readmitted on March 1, 1948, for a follow-up examination. She was in good health and had resumed duties as a female attendant at a Government hospital.

The following tests were performed to estimate the unction of the pancreas (W. Ratnavale).—

Glucose-tolerance Test (March 5).—

	Blood-sugar	*Urine Sugar*
Fasting sugar	96 mg.	Nil
½ hour after glucose	133 mg.	Nil
1 hour after glucose	182 mg.	Trace
1½ hours after glucose	136 mg.	Nil
2 hours after glucose	100 mg.	Nil

Examination of Fæces.—Hookworm ova +. Muscle fibre remnants have completely lost their striations. Protein digestion is apparently unimpaired.

Fat Examination.—

Neutral unsplit fat	34·1	Percentage of dried fæces
Split fat	10·72	
Total fat	44·82	

The total fat is increased (normal 33 per cent) and the percentage of unsplit fat is much higher than the normal, indicative of impairment of lipase action.

FIG. 138.—Photomicrograph of cyst wall in *Case* 1. The columnar mucinous cells lining long crypts are well shown. (× 48.)

Commentary.—A large unilocular pancreatic cyst was observed when the lesion was exposed at operation. The cyst was not recognized as a proliferation cyst until after it was opened, when smaller cysts were noted in the thickness of its wall. Despite this, the feasibility of enucleation had been considered as the lesser sac was patent and the cyst was loosely covered by the peritoneum rising off the posterior wall of the lesser sac. This case illustrates the desirability of attempting enucleation in all cases, the attempt being abandoned if adhesions make the dissection hazardous.

Case 2.—

HISTORY.—M. K., male Sinhalese, aged 8, was admitted to the General Hospital, Colombo, on June 19, 1948. The child had been seized with a very severe cramp-like abdominal pain, which caused him to shriek in agony, bend down and compress his abdominal wall in a vain attempt to obtain relief. The pain continued at this intensity for three days. Vomiting set in after a few hours, and the abdomen became distended. By the third day the vomiting had ceased and the pains now came on in bouts four or five times a day. At this

stage the boy had fever. The fever abated on the ninth day, but the bouts of abdominal pain continued for three weeks, coming on at longer and longer intervals. At this time, a visible bulging of the upper abdomen was noticeable, and this gradually increased so much in size that in the last week there had been difficulty in breathing after meals.

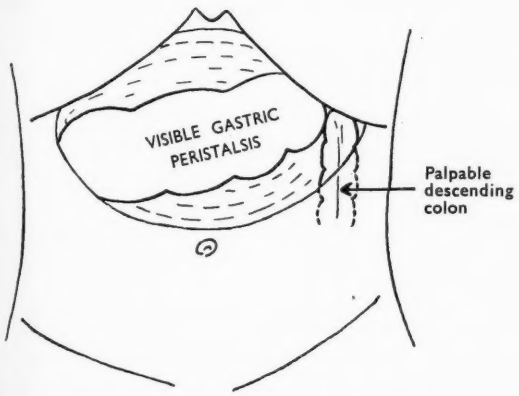

FIG. 139.—A diagram summarizing the principal physical signs in *Case* 2.

ON EXAMINATION.—A large round tumour was seen and felt in the upper abdomen extending down to the umbilicus. It did not move on respiration. It could be moved from side to side but not up and down. The descending colon was palpable as it ran down the left side of the tumour. The tumour was dull on percussion, except for a transverse band in front of it. This area was resonant on percussion and splashing could be elicited on palpation over this place. Visible peristalsis passing from left to right was seen over this area.

The physical signs were those of an upper abdominal round tumour lying behind the stomach and with the descending colon running down its left side (*Figs.* 139–141). The history of the case suggested that the tumour was a pseudo-pancreatic cyst following acute pancreatitis. Operation was advised on account of the distress produced by the bulk of the cyst.

AT OPERATION (July 2).—Under intratracheal anæsthesia, the abdominal cavity was opened by a left upper abdominal paramedian incision. A large cyst lay below the liver, and extended up to the left cupola of the diaphragm. The lesser omentum was tense and convex and the fluid in the cyst was visible through it. The stomach and the transverse colon lay in front of the cyst (*Fig.* 143), and the cyst bulged against the transverse mesocolon, which was laden with fat. A cautious attempt was made

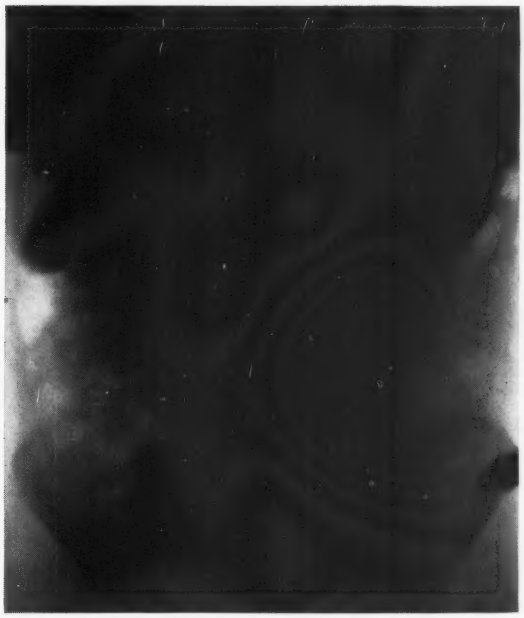

FIG. 140.—A radiograph taken immediately after a barium meal on June 30, 1948, in *Case* 2. The large filling defect produced by the projection of the cyst on the posterior gastric wall is shown.

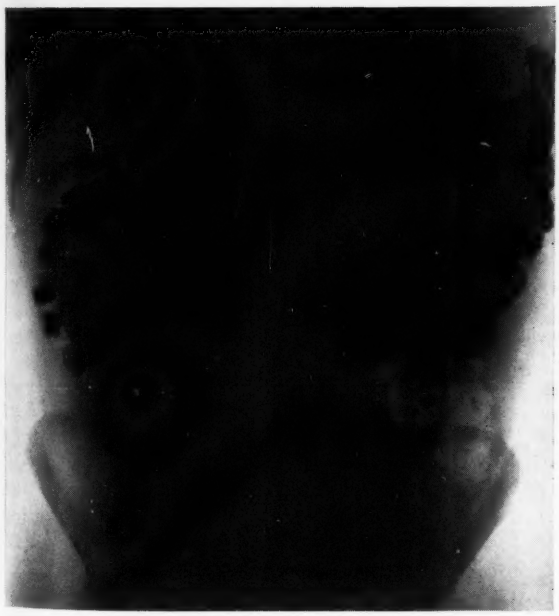

FIG. 141.—A radiograph taken 5 hours after a barium meal on June 30, 1948, in *Case* 2. The relations of the cyst to the colon are seen.

FIG. 142.—A radiograph taken half an hour after a barium meal on July 20, 1948 following operation in *Case* 2. The barium has entered the stomach, duodenum, and small intestines, but there is no trace of it in the cyst cavity.

to separate the cyst wall from the stomach through an incision in the gastrocolic ligaments, but the freeing of adhesions caused so much bleeding that the attempt was not persevered with. A Jurasz operation was decided on. The anterior gastric wall was opened by an incision 4 in. long in the long axis of the stomach, and the cyst was opened by an incision 3 in. long through the posterior gastric wall. A pale serous fluid flowed out. The cyst was emptied by an electric sucker. The cyst wall was

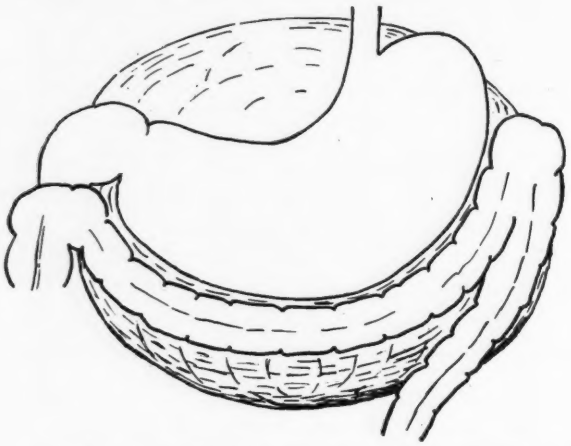

FIG. 143.—The relations of the cyst as seen at operation in *Case* 2.

⅛ in. thick and it was well defined, with patches of calcification in its posterior wall. A fresh attempt was made to separate the cyst wall from the stomach, but the bleeding proved to be as profuse as before and the attempt was abandoned. The edges of the incision in the cyst wall were united to the posterior gastric wall incision by a running over-and-over stitch, and the anterior gastric wall was closed by suture. At this stage the child was noted to be in severe respiratory distress. Pulse was 160 and of good volume. His colour was good. The respiratory distress passed off in the next half hour, but the child was restless for the next few days.

Apart from this complication, the child made an uninterrupted recovery, his wound healed by first intention, and the cystic tumour disappeared (*Fig.* 142). He was kept in hospital till Aug. 4 to permit of fuller investigation of his pancreatic functions.

REPORT ON CYST FLUID (Dr. N. G. Baptist, Lecturer in Biochemistry, University of Ceylon).—Solid matter 2·0 g. per 100 c.c. All protein colour tests were positive. No reducing carbohydrates and only traces of fat. Total nitrogen 0·6 mg. per cent. Enzymic activity: A dextrinogenic amylase was present. Slight lipolytic and proteolytic activity was shown. Trypsin appeared to be absent. In general the enzymic activity was feeble. No autodigestion was produced at 37° C. Conclusion: The fluid appears to be a weak solution of albumin and globulin with feeble enzymic activity.

OTHER TESTS (Dr. W. Ratnavale, Pathologist, General Hospital).—

July 1: Diastase index of urine 133·3.

Aug. 3: Examination of stools:—

Total fat	17·3	g. per cent
Split fat	6·163	g. per cent
Unsplit fat	11·136	g. per cent
Percentage of split fat to total	35·63	
Percentage of unsplit fat to total	64·37	

Report on a piece of meat swallowed in gauze bag and examined after passing stools: Muscle-fibres showed no nuclei or striations. Protein digestion appears to be satisfactory.

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EXPERIMENTAL SURGERY

POLYTHENE TUBING IN THE EXPERIMENTAL SURGERY OF THE URETER

By J. P. HERDMAN

FROM THE NUFFIELD INSTITUTE FOR MEDICAL RESEARCH, OXFORD

THE treatment of injuries of the ureter has always been unsatisfactory. In those cases in which it is possible to restore the continuity of the urinary tract by a uretero-ureteric or a uretero-vesical anastomosis, stenosis of the junction and hydronephrosis often develop (Bump and Crowe, 1929); while in the cases where an anastomosis cannot be performed, nephrectomy or a ureterocolic transplant is necessary. It would therefore be very helpful if some means could be found of bridging the gap between the ends of a cut ureter without subsequent production of a stricture.

It seemed that it might be possible to use for this purpose one of the newer plastics which have recently come into prominence, and which are chemically and biologically more inert than any substances previously available. Of these polythene appeared the most suitable. It is a transparent flexible solid of considerable tensile strength, which produces very little tissue reaction, and has water-repellent properties and an anticoagulant action on blood. A full description of its properties has been given by Farquar and Lewis (1948).

Polythene has been used successfully in animals to replace segments of the trachea, pelvic colon, and bile-duct (Brown, Grindley, and Craig, 1947). In the common bile-duct of dogs polythene tubes have been inserted for periods of up to six months without precipitation of organic or inorganic matter, and these tubes are now being used experimentally in the surgery of the human bile-duct (Walter, Grey, and Priestley, 1948). It seemed possible that, if polythene was suitable for permanent implantation in the biliary tract, it might also be suitable for use in the ureter. An investigation into this possibility has accordingly been carried out.

Rabbits, anæsthetized with nembutal and ether, were used. The polythene tubes were approximately 1 cm. long, of 1 mm. or 0·5 mm. internal diameter, with walls 0·25 mm. thick. It can be shown on theoretical grounds from Poiseuille's formula that a tube of these dimensions would offer a negligible mechanical resistance to the flow of urine. In five rabbits the ureter was approached through a lumbar incision and divided. A polythene tube was inserted into the two cut ends, and secured with four thread ligatures, two being tied around the proximal part of the ureter and tube, and two around the distal ureter and tube. In two of the rabbits this procedure was carried out on both sides and in three on one side only. In a further six rabbits the bladder was opened through a midline incision and a length of tube passed up through the ureteric orifice on one or both sides. The ureter was then exposed outside the bladder and ligatures passed around that portion of it which contained the tube. The ligatures were then tied around both ureter and tube, and the ureter sectioned between the ligatures, care being taken not to injure the underlying tube which now joined the two cut ends of the ureter.

The fate of these eleven animals is shown in *Table I* (No. 1–11). In all but one, which died four days after operation as the result of peritonitis,

Table I.—RESULTS OF THE INTRODUCTION OF POLYTHENE TUBING INTO RABBITS' URETERS

No.	Sides Operated On	Fate	Survival Time in Days	Post-mortem Findings
1	R.	Killed	67	Right pyonephrosis
2	R.L.	Died from leak from ureter	3	Distended renal pelves and ureters on both sides
3	R.L.	Died from ? uræmia	18	Right hydronephrosis; left pyonephrosis
4	L.	Killed	5	Early left hydronephrotic changes
5	L.	Died from peritonitis	4	Left kidney macroscopically and histologically normal
6	R.L.	Died from uræmia	6	Early bilateral hydronephrotic changes
7	R.L.	Died from leak from bladder	3	Distended renal pelves and ureters on both sides
8	R.	Killed	26	Large right hydronephrosis and hydroureter
9	R.	Killed	20	Right hydronephrosis and hydro-ureter
10	L.	Killed	42	Left hydronephrosis
11	L.	Killed	11	Left hydronephrosis and hydro-ureter
12	R.	Killed	10	Left hydronephrosis and hydro-ureter
13	L.	Killed	15	Left hydronephrosis and hydro-ureter
14	R.	Killed	15	Right hydronephrosis and hydro-ureter
15	L.	Killed	7	Left hydronephrosis

there was evidence of urinary obstruction both macroscopically and histologically. This varied in degree from distension of the renal pelvis and ureter with flattening of the renal papilla in the early cases, to a well-developed hydronephrosis and hydroureter in the later ones (*Fig.* 144). Microscopical examination of the kidneys showed the general features of hydronephrosis that other workers have obtained by ligation of the rabbit ureter (Hinman, 1945; Strong, 1940). The tube and ureter appeared to be firmly united in those animals which survived a week or more.

There were two possible explanations of these results. The tube might have become blocked with urinary deposits: or the obstruction might have been a functional one, due to interruption of the neuromuscular continuity of the ureter, and

consequent interference with peristalsis in the distal segment, of the type which has been postulated by various authors (Bump and Crowe, 1929). Microscopical examination of the tubes, however, showed that they were full of urinary deposits (*Fig.* 145), and attempts to inject fluid through them by means of a hypodermic syringe revealed evidence of obstruction. This obstruction was therefore mechanical rather than functional in nature. To confirm this, polythene tubes were introduced into the ureters of four more animals. The tubes were passed up through the ureteric orifice, and secured by ligatures as before, but on this occasion the lower ends were left projecting into the bladder. In this way no part of the ureter was left distal to the tube and any possibility of failure of peristalsis in an isolated distal segment was eliminated. In all four animals urine could be seen entering the bladder in peristaltic jets in the normal manner at the end of the operation. Nevertheless, these animals developed hydronephrotic changes in exactly the same way as the others (*Table I*, No. 12–15). It is clear that the obstruction is a mechanical one due to the precipitation of urinary deposits on the tube and is not caused by a disturbance of ureteric peristalsis.

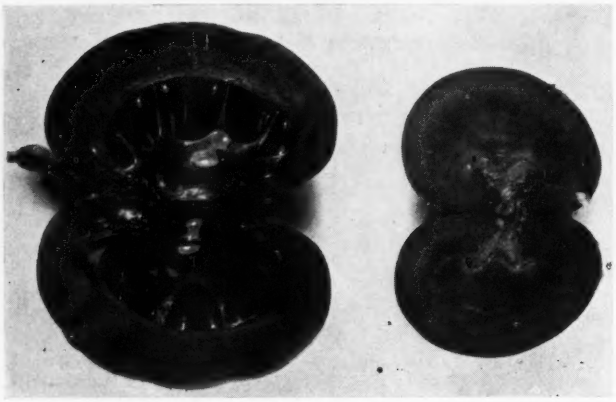

FIG. 144.—Kidney after insertion of polythene tube in ureter for 26 days (A), with normal kidney (B) for comparison. Both kidneys have had post-mortem intra-arterial injections of 2 per cent Berlin blue solution.

It is of interest that in no case did a primary renal atrophy occur. This observation is in accordance with the findings of Hinman (1934) that obstruction of the ureters of experimental animals, whether partial or complete, is followed by hydronephrosis and not by primary atrophy. Although there is no satisfactory evidence that man behaves differently from other animals in this respect, the view that ligation of the human ureter is usually followed by atrophy of the kidney is still held by some writers.

These results show that the introduction of polythene, despite its inert properties, leads to the precipitation of urinary deposits in the same way as other foreign materials do. While it seems unlikely that the difference in the composition of the urine and the size of the ureter in man would influence this result appreciably, any final decision must await investigations carried out in larger animals. Meanwhile, it can be concluded provisionally that polythene tubing is unsuitable for permanent implantation in the ureter.

SUMMARY

Polythene tubes were introduced into the ureters of rabbits. This led to hydronephrotic changes as a result of the rapid blockage of the tubes by urinary deposits. Primary renal atrophy did not occur in any animal.

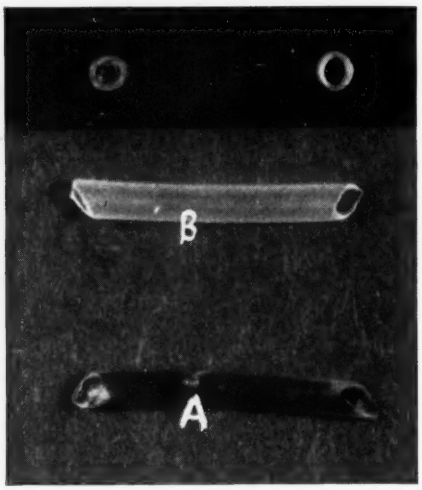

FIG. 145.—Polythene tube removed from ureter after 42 days (A), with normal tube for comparison (B), together with cross-sections of the two tubes. (Magnification × 2.)

My thanks are due to Dr. N. T. Jaco, of the Department of Pathology, The Radcliffe Infirmary, who undertook the histological examinations; and to Dr. G. S. Dawes, Director of the Nuffield Institute for Medical Research, for advice in the preparation of this paper.

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SHORT NOTES OF RARE OR OBSCURE CASES

LARGE SACCULAR ANEURYSM OF THE INTRACRANIAL PART OF THE VERTEBRAL ARTERY

By A. N. GUTHKELCH, Manchester

The following case appears to be of sufficient rarity and clinico-pathological interest to warrant publication.

CASE REPORT

History.—Mrs. C. H., a housewife aged 32, was admitted to Salford Royal Hospital on Oct. 10, 1947, complaining of headaches, clumsiness of the limbs, and slurred speech. Her history went back to 1936 when she was admitted to a Fever Hospital with what she had since been told was 'suspected meningitis'. As she was transferred within the next day or two to the Neurological Unit of a General Hospital she had probably (as we now believe) suffered a subarachnoid hæmorrhage, but unfortunately the case notes relating to this illness could never be traced.

When she recovered she found that she was completely deaf in the left ear, but otherwise, except for occasional headaches of short duration, she remained well. Towards the end of 1944 she began to notice the gradual onset of clumsiness of the hands and difficulty in walking. At about the same time her voice began to get weak and hoarse. However, she did not seek medical advice until six weeks before admission, when she had a sudden attack of vomiting, from which time until her admission to hospital she had been confined to bed by severe dizziness which increased immediately she tried to raise her head from the pillow. During these weeks, however, there was no increase in her headaches.

On Examination.—The abnormalities found were restricted to the central nervous system; the blood-pressure was 124/80 mm. Hg.

There was no neck stiffness and the optic disks were normal. There was coarse cerebellar nystagmus, more prominent to the left than to the right, a diminished left corneal reflex, slight left facial weakness, and complete nerve-deafness in the left ear. The left side of the palate and the left vocal cord were paralysed, the voice being weak and the speech slurred and unrhythmical. The left arm and leg were grossly hypotonic and ataxic, the right somewhat less so.

X-ray examination of the skull was negative. Lumbar puncture showed a clear colourless fluid under 140 mm. pressure, with no block to Queckenstedt's test. The total protein of the C.S.F. was 74 mg. per cent, with increased globulin and 3 cells per c.mm. The Wassermann reaction was negative in blood and C.S.F.

The pre-operative diagnosis was one of tumour of the left cerebellopontine angle. Acoustic neuroma appeared to be excluded by the relatively low C.S.F. protein and the absence of radiological changes, but although we were inclined to associate the present symptoms with the illness in 1936—of which we really knew very little—we did not feel at all sure of their exact pathological cause.

At Operation (Nov. 22).—A left-sided cerebellar exploration was made. The dura was pulsating normally, without any sign of increased tension, and on opening it we found the cisterna magna to be of normal size. But above this there was a huge, greenish-black, hard, irregular, somewhat nodular mass extending deep into the left cerebellopontine angle. The left vertebral artery entered its lower pole, the left lobe of the cerebellum was reduced to little more than a flat cap over it, and the medulla was distorted into a thin triangular strip wrapped around its posteromedial aspect. The wall of the aneurysmal sac—for such it evidently was—proved to be very thick and little pulsation could be felt within it, but any attempt at mobilization produced immediate severe respiratory embarrassment. Eventually it was decided that the vertebral artery should be ligated in an attempt to prevent further expansion of the aneurysm, and this having been done the operation was terminated.

The patient regained consciousness immediately after operation, but her blood-pressure began to mount steadily and she died of acute respiratory failure six hours later.

At Autopsy.—The brain was removed entire and fixed in formalin; the specimen is now in the Museum of the Manchester University Department of Pathology. The aneurysm proved to measure $3 \times 3\frac{1}{2} \times 4$ cm. The drawings reproduced here (*Fig.* 146) show the extreme degree of flattening of the medulla and the way in which it has been so thrust to one side by the aneurysmal sac that the right vertebral artery appears to be running on the left side of the ventral aspect of the medulla, while the right posterior inferior cerebellar artery almost encircles it. Superiorly the left cerebellar hemisphere and left side of the pons have been greatly compressed. The trigeminal nerve emerges between the aneurysmal wall and the pons, while the facial and auditory nerves, which are not shown in the drawings, crossed anterior to its upper pole by which they were greatly flattened. The glossopharyngeal and vagus nerves (also not shown) ran into the medial wall of the inferior part of the sac, from the lateral aspect of which they only emerged at their point of exit from the cranial cavity.

There was no internal hydrocephalus.

COMMENT

Dandy (1944) refers to a number of recorded cases of large saccular aneurysm of the vertebral artery, and adds 2 of his own. His first case is strikingly similar to that recorded here, but with the difference that in his case the aneurysm arose from one side of the vertebral artery, so that he was actually able to shell out the whole mass, though with fatal issue. Moreover in both Dandy's cases the intracranial pressure was extremely high.

The complete absence of any increase in intracranial pressure in the present case is remarkable. It is of course well known that space-occupying lesions of the posterior fossa may attain a large size without causing papillœdema and occasionally without increase in the lumbar or even the intraventricular pressure; but in this case there was not even any diminution in the size of the cisterna magna. It is tempting to suggest that the direction of the flow of blood throughout the sac resulted in its

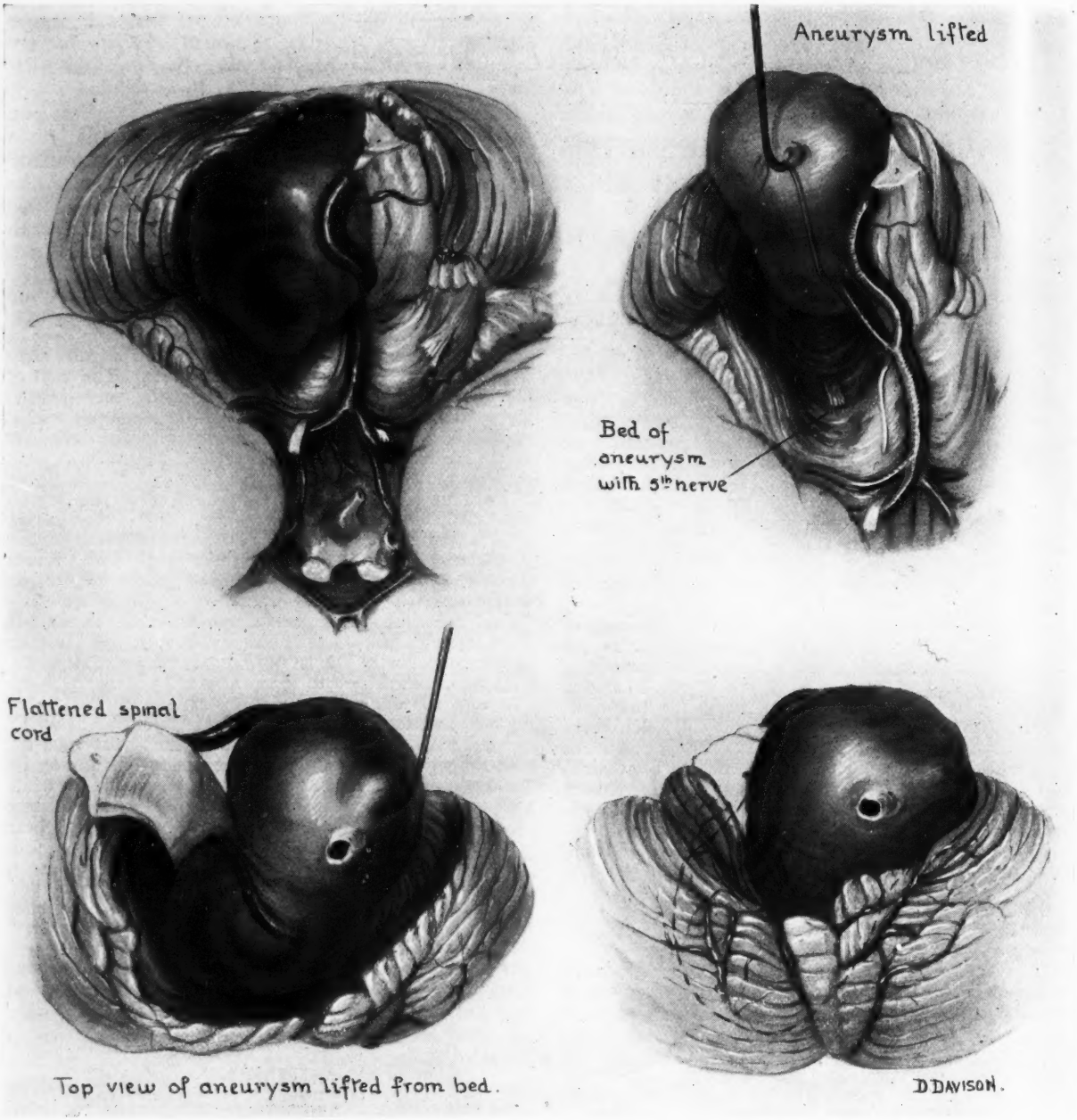

FIG. 146.—Various drawings of the aneurysm made at autopsy. Note the position of the right vertebral artery in relation to the medulla.

pulsating always in an upward direction, so as to hammer away the cerebellum and pons as it advanced, and that the rate of advance was so slow that any significant increase in the size of the aneurysm was always compensated by an equal degree of destruction of brain substance. The fact that in the present case the symptoms were of several years' duration, whereas in Dandy's they were 8 and 12 months respectively, tends to support this explanation.

My grateful thanks are due to Dr. Magnus L. Thomson, who originally referred this case to me; and to Miss D. Davison for the drawings of the autopsy specimen.

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A CASE OF COMPOUND INTUSSUSCEPTION OF A MUCOCELE OF THE APPENDIX

By J. N. WARD-McQUAID, Oxford

Intussusception of the appendix is rare, and only 7 cases of intussusception of a mucocele of the appendix have been traced. Six of these were reviewed by Fraser (1943), and 1 further case, full details of which are not available, was reported by Uriburu (1946).

A mucocele of the appendix may be complicated by rupture, giving rise to pseudomyxoma peritonei, by adhesion to neighbouring structures causing intestinal obstruction, by torsion, by concretion formation, and finally by intussusception.

An intussusception of an appendix mucocele may be partially or completely into the cæcum, or it may be compound in character, the mucocele taking with it both cæcum and ileum into the ascending colon. A case of compound intussusception of a mucocele of the appendix is now described.

CASE REPORT

History.—R. R., aged 57 (R. I. No. 90090/48), a domestic servant, was admitted to the Radcliffe Infirmary as an emergency on July 15, 1948, complaining of abdominal pain, vomiting, and constipation.

In the last year her habitual constipation had increased and had been associated with rectal bleeding on straining and loss of weight. A month previously, while in the

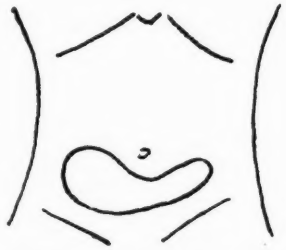

Fig. 147.—Diagram of the palpable abdominal tumour on the day of admission.

cinema, she had been seized with a severe generalized abdominal colic, lasting only three minutes, but followed by retching and vomiting for three hours. Later she had two similar but less severe attacks. Five days before admission she was again doubled up with severe abdominal colic. The pains lasted two minutes, with five minutes' interval at first, and although mainly central, were also felt in the right iliac fossa and in the small of her back. Two days later, the patient went to work feeling well between residual spasms of pain, which were made worse by eating and relieved by lying down. At the time of admission her bowels had not been open for a week, and she had last vomited the previous day. There had been no symptoms suggestive of a previous attack of appendicitis, and the family history was not relevant.

On Examination.—T. 99° F. (37·3° C.), P. 90. Her tongue was furred and fœtor was pronounced. The abdomen contained a firm tender, mobile, comma-shaped, mass, some 20 cm. long and varying in width from 2·5 cm. to 7·5 cm., which extended from the right of the umbilicus across to the left hypogastrium (*Fig.* 147). The mass was dull to percussion and moved slightly on respiration. The whole right iliac region was very tender. On rectal examination there was blood on the examining finger after withdrawal. Apart from a small irreducible para-umbilical hernia and a blood-pressure of 200/120, no other abnormality was found.

The following day her symptoms had subsided and the physical signs had changed. There remained a smaller

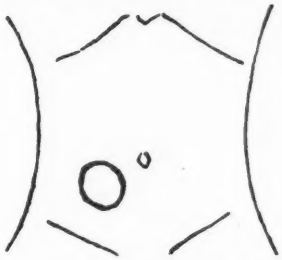

Fig. 148.—Diagram of the palpable abdominal tumour on the following day.

and less defined tender mass, about 4 cm. in diameter, below and to the right of the umbilicus (*Fig.* 148).

Radiological Reports.—On admission a straight radiograph showed no fluid level. Later a barium enema

Fig. 149.—Radiograph of the barium enema, showing the rounded filling defect in the cæcum at the base of the appendix.

(*Fig.* 149) was reported on by Dr. F. H. Kemp as follows: "There is a tumour mass in the cæcum. Precise nature not determined."

Progress.—She had three short attacks of colic, but there was no vomiting and she felt well in the intervals.

The small abdominal mass remained. Laparotomy was decided upon with the provisional diagnosis of carcinoma of the cæcum.

AT OPERATION.—On July 28, under general anæsthesia, the abdomen was explored by Mr. A. Elliot-Smith through a right lower paramedian incision. A compound intussusception of ileum, cæcum, and appendix into the ascending colon and extending to the region of the hepatic flexure was discovered and easily reduced. The

FIG. 150.—Photograph of the hemicolectomy specimen, showing the appendix mucocele projecting into the cæcum.

appendix was then seen to be a mucocele and its base felt continuous with a firm rounded mass inside the cæcum. The cæcum and the ascending colon were mobile. There was one firm gland in the mesentery along the ileocolic vessels, and the liver and pelvis were normal. A right hemicolectomy was performed with end-to-side anastomosis, and the abdomen closed.

Convalescence was uneventful and she was discharged on the thirteenth post-operative day.

SPECIMEN (R.I.S.H. 3234/48) (*Fig.* 150).—The appendix was entirely converted into a mucocele, the bulbous proximal end being partially intussuscepted into the cæcum and the cæcum inverted 0·5 cm. on itself. The rounded swelling projected 2 cm. into the cæcum, and where the cæcum folded on itself there was a small area of ulceration. The lumen of the cæcum did not communicate with the mucocele. There was no evidence of malignancy on histological examination.

COMMENT

Intussusception of the appendix is characterized by recurrent short, but severe, attacks of central abdominal colic, associated with pain in the right iliac fossa and vomiting, with intervals of complete freedom from symptoms. There is often pain in the back, loss of weight, pain on defæcation, and occasionally passage of blood by the rectum. When the intussusception becomes compound, symptoms of subacute obstruction are found, together with a palpable mass in the right iliac region. Such a history, except perhaps for the right iliac fossa pain, could be said to be that of any intussusception in the ileocæcal region. A barium enema may indicate its origin by showing filling of the ileum together with a rounded filling defect in the cæcum caused by the base of the intussuscepted appendix. A similar clinical and radiological picture can be presented by an early carcinoma arising near the base of the appendix, and forming the apex of an intussusception. Even at operation its exclusion may be difficult.

In the case reported, had it been possible to recognize the nature of the condition at operation, and to exclude carcinoma as the cause of both mucocele and intussusception, a local resection of the appendix mucocele with preservation of the ileocæcal valve would have been the appropriate treatment.

I am indebted to Mr. A. Elliot-Smith for permission to publish this case, and to Dr. F. H. Kemp for the radiograph.

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INTESTINAL OBSTRUCTION DUE TO ASCARIS

BY SAMUEL SKAPINKER
JOHANNESBURG, SOUTH AFRICA

INTESTINAL obstruction due to an impaction of round worms is not an infrequent occurrence in the Bantu in South Africa. It is difficult to state the incidence, but it is more common than malignancy of the bowel as a cause of obstruction. The Bantu is less prone to intestinal carcinoma than the white population.

A very high percentage of the Bantu are infected by worms and the ascaris is the most common parasite.

This case is being presented for its most unusual X-ray picture.

CASE REPORT

CLINICAL HISTORY.—The patient, 5885, S. T., a Bantu male aged 26 years, was admitted to the second surgical unit of Baragwanath Hospital on Aug. 13, 1948. His history was that on Aug. 10 he began to have a continuous pulling abdominal pain, centred around his umbilicus. He began to vomit that day and for the next three days he vomited about six times daily. Constipation was present since the onset of pain until the day of admission, when he had a small bowel motion.

ON EXAMINATION.—The patient was a young adult male obviously in pain. His eyes were sunken, but his

tongue was moist. His cardiovascular and respiratory systems revealed nothing abnormal.

His abdomen was slightly distended and moved freely on respiration. There was no visible peristalsis. Palpation revealed several masses, two of which were very distinct, very tender, and mobile. These two were round and hard and were to the left and right of

FIG. 151.—Radiograph showing (A) dilated distended loop of bowel containing two worms.

the umbilicus. The borborygmi were increased. Rectal examination revealed a tense mass in the rectovesical pouch.

Scout Film taken with the Patient Supine.—As can be seen in this radiograph, there is a small-bowel obstruction with numerous fluid levels. In a dilated loop of small intestine (marked A) two round worms can be plainly seen. Several masses of impacted worms can be seen to the right and the left of the umbilicus. From this radiograph, a diagnosis of intestinal obstruction due to round worms was made (*Fig.* 151).

TREATMENT.—The patient was placed on continuous duodenal syphonage and his hydration was maintained by intravenous fluid therapy. There was no relief of the obstruction after forty-eight hours of conservative treatment and a laparotomy was performed under cyclopropane and curare anæsthesia. Two main sites of obstruction were found, one in the ileum and the second in the jejunum.

An enterotomy was performed and several hundred round worms were removed. The bowel was sutured with silk.

PROGRESS.—Except for an anaphylactic reaction that the patient had post-operatively, the convalescence was uneventful and the patient was discharged home on Sept. 5.

He was seen one month later at Out-patients, when he was feeling fit, and given an anthelmintic to clear him of the remaining worms.

DISCUSSION

This case is unusual in that the worms could actually be seen on the X-ray picture in the dilated loop of jejunum and gave the diagnosis.

Archer and Pietersen (1930), and before them Fritz (1922), both used barium as a contrast medium to show up the ascaris, but I cannot find a record of worms being demonstrated on a flat film.

I must thank the Departments of Medicine and Surgery of the University of Witwatersrand Medical School for the help in reproducing this radiograph.

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AN ADVANCED CASE OF MEGA-ŒSOPHAGUS DUE TO CARDIOSPASM

BY STANLEY O. AYLETT, LONDON

CASE REPORT

THE patient, an old lady of 84, with well-preserved mentality, complained of great difficulty in swallowing. The difficulty had existed for about twenty years, although the insidiousness of its onset did not enable the patient to place an exact date on its commencement. She remembered that in September, 1939, she had to give up eating meat, as for a long time she had had marked difficulty in swallowing this type of food and finally could manage it no longer, even when finely cut. She went on to a diet of steamed fish, soft foods, and milk and has existed on this until recently.

A few weeks prior to admission to hospital, the swallowing had worsened. She was now living on a few cups of milk a day with an occasional biscuit soaked in the fluid.

There had been marked loss of weight and increasing weakness, the latter making her almost bedridden. Swallowing recently could only be achieved by constant repetition of the act with each sip of fluid that was taken. Often it was followed by paroxysmal attacks of coughing and regurgitation of the fluid, together with a quantity of frothy mucus.

The patient was aware of a swelling in her neck, but was uncertain when this had commenced. She had had no illnesses of note apart from the present one, and her life had been that of the normal hard-working housewife from the East End of London.

CONDITION ON ADMISSION.—A frail old lady, showing signs of gross inanition and dehydration. Weight, 5 st. 5 lb. Her most noteworthy feature was a large swelling in the neck, extending up as far as the hyoid bone and disappearing downwards into the thoracic inlet (*Fig.* 152). At first sight the swelling gave to the patient's neck the appearances of a large colloid goitre. The swelling, soft but not easily compressible, lay, however, behind the larynx and upper trachea, both of which were displaced forwards. It was noticed that when the patient attempted to drink, the forced efforts necessary to achieve this act were accompanied by air swallowing, with resulting increase in the size of the swelling in the neck.

Apart from chronic bronchitis and a femoral hernia, other systems were well within the limits of normal for the age of the patient.

A barium swallow confirmed the clinical diagnosis of advanced œsophageal dilatation due to cardiospasm (*Fig.* 153).

A B

FIG. 152.—A, B, Showing the marked swelling of the neck due to dilatation of the cervical œsophagus.

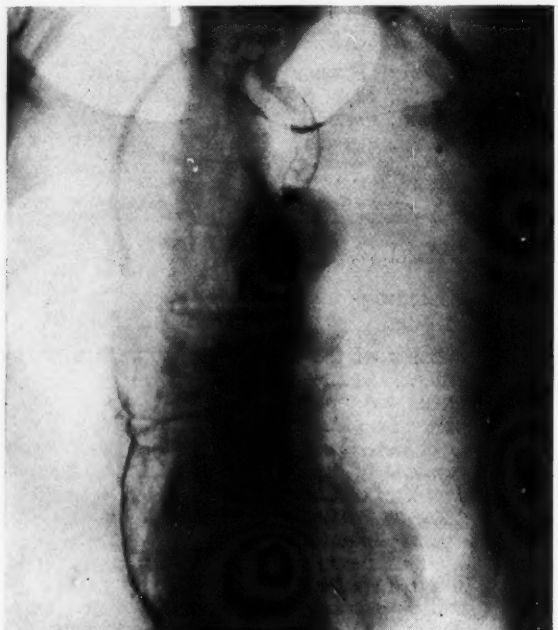

FIG. 153.—Radiograph showing the dilated cervical portion of the œsophagus.

TREATMENT AND PROGRESS.—In order to improve her general condition it was decided to feed her by a continuous drip method. What was quite remarkable was the comparative ease with which it was possible to introduce the Ryle's tube. She remained on the drip for a week, being given a high calorie diet, supplemented by vitamins and protein extracts. At the end of that time dilatation with mercury bougies was commenced. The patient was highly co-operative and soon learned to pass them three times a day herself. With this treatment she was able to take a semi-solid diet of over two thousand calories each day. At her request she was discharged at the end of a month's treatment.

Three weeks later the patient was readmitted with a strangulated femoral hernia. She survived operation satisfactorily and during her convalescence was eating ordinary ward diet, including meat cut into small portions. The swelling in her neck was less, probably because she was no longer air swallowing, but a further radiograph revealed a similar dilatation of the œsophagus. Her weight on discharge was just over 6 st.

Comment.—Such a degree of cervical œsophageal dilatation is rare. Apart from a case described by H. Edwards, I have not been able to locate the description of another. Much of the dilatation was, I believe, due to constant dilatation of the œsophagus by swallowed air. Only by this aerography was the patient able to force food down, and this added to the distension already present.

The ease with which the very light Ryle's tube passed through the cardio-œsophageal junction into the stomach has been noted. Why the weight of a few mouthfuls of fluid should not similarly overcome any resistance at this level is difficult to explain. It would seem that in advanced cases such as this there is interference, not only with the cardio-œsophageal junction, but with the whole mechanism and co-ordination of the complicated act of swallowing.

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A CASE OF GASTROLITHS

By R. C. ALEXANDER, C.B.E.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF ST. ANDREWS AND ROYAL INFIRMARY, DUNDEE

CASE REPORT

G. T., male, aged 62, employed as a loader in the goods yard, was referred to me at Dundee Royal Infirmary by Dr. J. S. Y. Rogers, Dundee, in the spring of 1940, complaining that for three years he had vomited after foods. In the two months previous to examination, milk, milk puddings, and lean meat caused vomiting also. Until 1937 he had enjoyed excellent health and had a good appetite, but since then his appetite had diminished and he had lost 3 st. in weight, but had only recently given up work.

FIG. 154.—Radiograph taken five hours after barium meal. (April, 1940.)

FIG. 155.—A later radiograph, twenty-four hours after barium meal. (April, 1940.)

ON EXAMINATION.—There was no abdominal tenderness, but gastric splashing was elicited two hours after food.

Test-meal: Showed slight increase in free and combined acid, no blood or bile. Mucus was present throughout and starch especially in the third hour.

X-ray Examination: Large stomach, considerable narrowing of pylorus, delay in passage of barium meal, and one or more circular filling defects (*Figs.* 154, 155).

The provisional diagnosis was pyloric stenosis with multiple simple gastro-adenomata.

Gastroscopic examination was not possible.

TREATMENT.—On May 3, after gastric lavage, the abdomen was opened and several rounded bodies were palpated freely movable in the stomach. An incision was made in the anterior wall of the stomach and six foreign bodies were removed. An anterior gastro-jejunostomy was performed. As the clamps were being removed, it was discovered that two further bodies had been overlooked. One was removed by reopening the

FIG. 156.—Radiograph taken shortly after a barium meal. (August, 1948.)

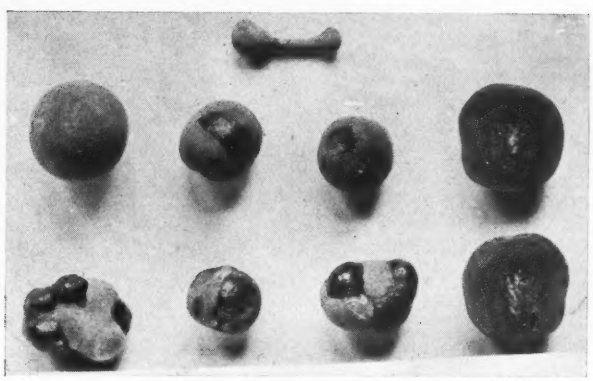


FIG. 157.—Photograph of the eight gastroliths, with one in section. ($\times \frac{1}{10}$.)

anastomosis, the other was passed into the jejunum and was found in the stool on the seventh day.

The patient was re-examined on Aug. 6, 1948. He has now retired from work, enjoys good health, and takes his food without discomfort.

Radiologist's report is as follows: "Œsophagus negative. Stomach showed a normally functioning gastro-enterostomy opening. No barium passed through the pylorus and beyond a slight thickening of the rugæ around the stoma, no abnormality was found" (*Fig.* 156).

THE APPEARANCE OF THE SPECIMENS.—These consist of eight bodies of irregular shape (*Fig.* 157). The total weight of the bodies is 47 g. The bodies are of hard consistence and very closely resemble enteroliths. Some barium was deposited on the surface and the black coloration was due to organized blood-clot. One of the bodies was sectioned and showed concentric laminæ around a central nucleus. The pathologist reported that the bodies contain hair and vegetable fibre. It is probable that alkaline salts, taken to relieve dyspeptic symptoms, formed a considerable proportion of the bulk, as there would be retention of these in the stomach consequent on the presence of pyloric stenosis.

SUMMARY

The formation of concretions ('gastroliths') in the stomach of a man aged 62 is discussed and the symptoms and treatment are described.

SECONDARY HÆMANGIOMA OF BONE

REPORT OF A CASE

BY J. A. WHITE

LATE SURGEON, PARK PREWETT HOSPITAL, BASINGSTOKE

IN the nineteenth century angiomatous conditions of bone were not regarded as extreme rarities. Undoubtedly, however, many cases of vascular osteogenic sarcoma, osteoclastoma, or Ewing's tumour were reported as angiomata.

Bucy and Capp (1930) reviewed the proved cases of primary angioma of bone. There were 47 cases, of which only 11 were of the extremities. Geshickter

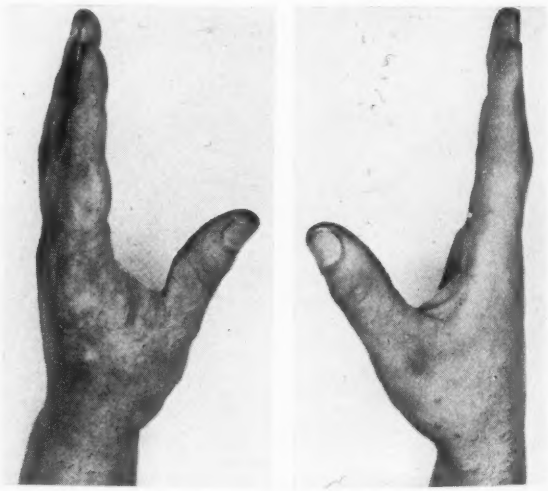
FIG. 158.—Photograph of the left shoulder and arm, showing the angioma.

and Maseritz (1938) added 16 proved cases from the literature and reported 4 more cases of their own of the extremities. They were careful to exclude a case with a skin angioma which probably involved a long bone secondarily.

Secondary hæmangioma of bone is associated with a similar contiguous soft-tissue condition, and has not achieved much prominence in the literature. It has been reported in association with severe angiomata (Couch, 1935) and with congenital arteriovenous fistulæ (Horton, 1932; White, 1946).

CASE REPORT

The patient, a healthy male aged 21, fractured his left radius and ulna in a car accident on June 19, 1945. He was treated by reduction and immobilization in plaster, but when seen on Dec. 8 there was clinical non-union of the radius with marked bowing. There was



A B

FIG. 159.—A, Photograph of the left hand to show the angioma; B, Photograph of the right hand for comparison of size.

also present an extensive skin and soft-tissue angioma extending from the left shoulder region to the left hand (*Fig.* 158.)

The patient stated that his 'birthmark' was first noticed when he was 10 months old, after he had fallen and broken his left clavicle. It slowly increased in size, and at the age of 7 he had two forms of treatment, carbon-dioxide snow and "electrical needles". As a boy of 13 it was noticed that his left hand was smaller than his

right (*Fig.* 159). In 1940 he fell and broke his left forearm, which united with slight angulation. In 1944 he was shovelling coal when he felt pain in the left forearm, and noticed increase in angulation. A radiograph, however, revealed no fracture. Every six to twelve months since he can remember, he has had pain in the left arm, with the formation of tender lumps, obviously due to attacks of thrombosis.

FURTHER EXAMINATION AND INVESTIGATIONS.—No murmur was heard over any part of the angioma. Blood-pressure in the affected arm was 134/86 mm. Hg, and in the normal arm 150/94. X-ray examination showed

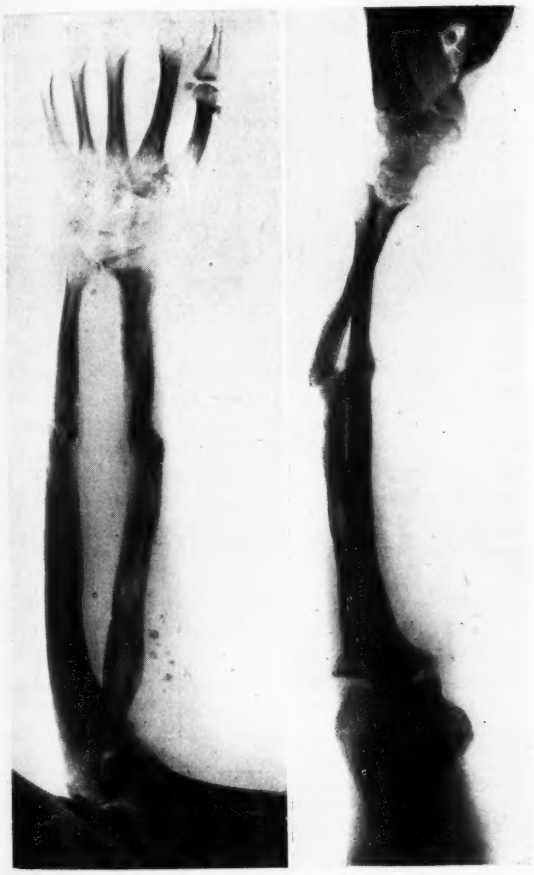


FIG. 160.—Radiograph, Dec. 8, 1945, showing united fracture of ulna, un-united and angulated radial fracture, angiomatous condition of radius and ulna, and soft-tissue phleboliths.

that the left humerus was slender and slightly shorter than the right. The left radius and ulna were also smaller than the right and extensively angiomatous (*Fig.* 160). This radiograph also shows a fracture of the radius, un-united with backward angulation, and numerous soft-tissue phleboliths. The bones of the left hand were smaller than the right. All other bones were normal, and no angiomatous changes were seen in any bones but the left radius and ulna.

Serum-calcium, phosphorus, and phosphatase were all within normal limits. No facilities were available for determination of venous oxygen content.

TREATMENT AND PROGRESS.—It was decided to graft the radius and at the same time remove a portion of bone for microscopy. On Dec. 18 an onlay graft, taken from the tibia, was fixed in position with four vitallium screws, after excising bone-ends and correcting the position. The bone was soft and vascular, with paper-thin cortex,

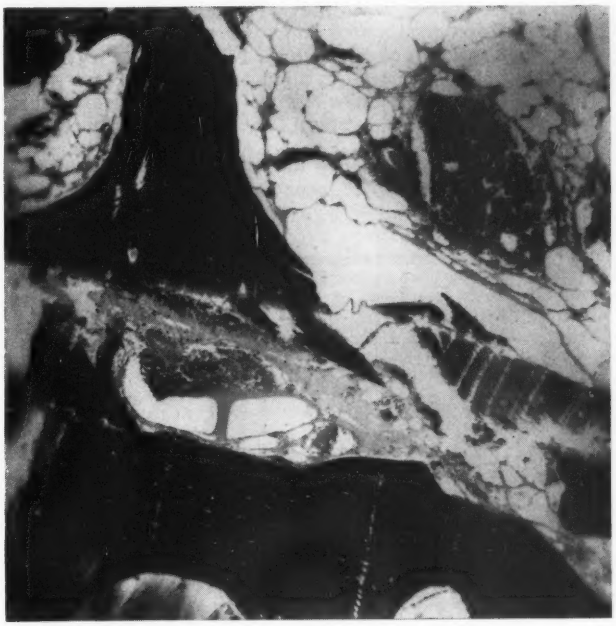


FIG. 161.—Microphotograph of section, showing bone invaded by angiomatous tissue. (× 90.)

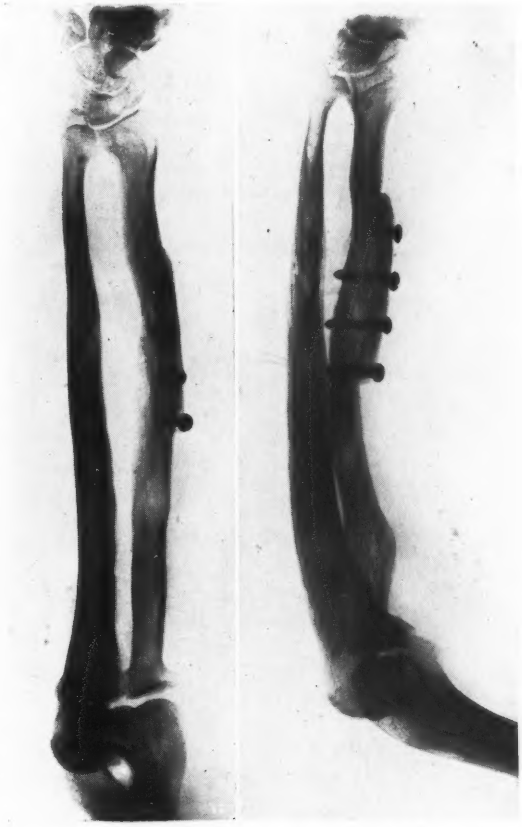


FIG. 162.—Radiograph, June 13, 1946, showing firm bony union following graft.

and cut very easily with bone forceps. A pneumatic tourniquet was used because of severe hæmorrhage. Report on the excised pieces of bone confirmed the diagnosis of hæmangioma (*Fig.* 161).

Convalescence was uneventful and *Fig.* 162 shows the condition six months later, union being complete.

The patient was re-examined on Dec. 4, 1947, two years after his bone-graft. He was working as a garage hand without difficulty, though supination was limited by about 40°. A radiograph (*Fig.* 163) shows firm union.

DISCUSSION

1. Treatment.—Complete excision of secondary bone hæmangiomata can rarely be possible apart from amputation. Deep X-ray therapy would probably be of value in the early growing stage, but not when growth of the tumour has ceased, as it does in adolescence. Fractures appear to be common owing to the pathological weakness of the bone, but usually unite readily. If malunion or non-union occurs it is possible to resort to open operation and bone-graft with success.

2. Differences between Primary and Secondary Long-bone Angiomata.—There are several obvious differences which may be briefly tabulated as follows :—

	Onset	Pain	Affected Limb	Progress	X-ray	Microscopical Appearances
Primary	Adolescent or early adult	Bone pain constant	Otherwise normal	Steadily progressive	Soap-bubble appearance. Sometimes bone expansion. No phleboliths	Little bone. Much connective tissue
Secondary	Present at birth or soon after	No bone pain. Pain in thromboses	Angiomata elsewhere often superficial. Limb may be larger or smaller	Stationary after adolescence	Fine longitudinal striation and trabeculation. No differentiation between cortex and medulla. Soft tissue phleboliths	Much bone. Little connective tissue

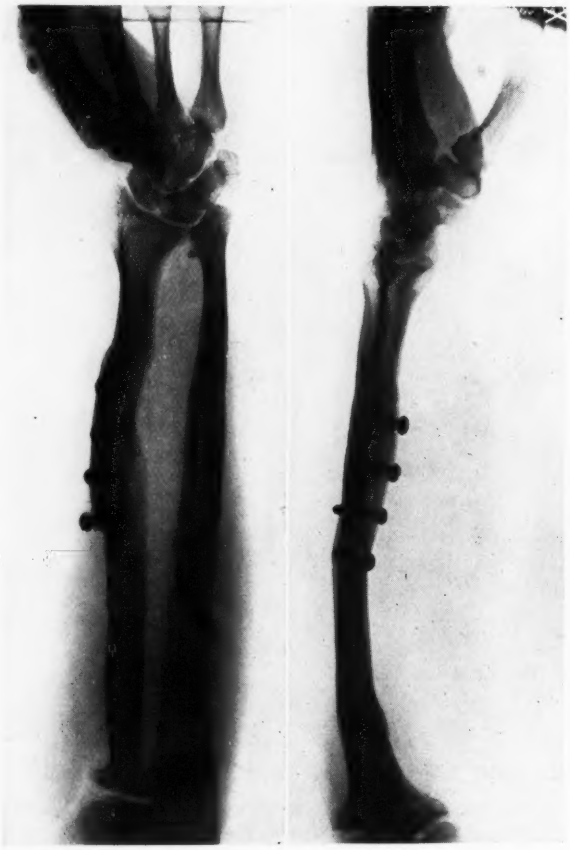


Fig. 163.—Radiograph, Dec. 5, 1947, two years after bone-grafting.

3. The Local Dwarfism.—This is in contrast to the local gigantism reported in cases of congenital arteriovenous fistulæ of the extremities (Horton, 1932 ; White, 1946). It must be due to a diminished blood-supply to the growing bone-ends, just as there must be increased blood-supply in gigantism. The following table compares the two conditions :—

	Present Case	Congenital Arteriovenous Fistula of Hand (White, 1946)
Condition	Cavernous hæmangioma	Multiple congenital arteriovenous fistulæ
Hand and arm	Smaller	Larger
Blood-pressures	Normal arm 150/94 Affected arm 134/86	Normal arm 95/60 Affected arm 124/40

Thus one condition causes a smaller limb with a lower blood-pressure and the other a larger limb with a higher blood-pressure. The former can be explained by the parasitic effect of the large angioma. The latter is not so easily explained. Arteriovenous fistulæ cause increased venous pressure which, if transmitted back to the heart, will cause tachycardia, cardiac hypertrophy, and some increase of general blood-pressure, but this does not account for the local increase of blood-pressure.

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NEURINOMA OF CERVICAL SYMPATHETIC CHAIN

By E. N. CALLUM, Ashford, Middlesex

The account of a neurinoma of the vagus nerve, reported by Murley in the July, 1948, number of the British Journal of Surgery, prompts the recording of the following case history.

CASE REPORT

Mrs. L. F., aged 31 years, came to hospital complaining of a lump in the right side of the neck which had started eighteen months ago, after an attack of tonsillitis.

On examination there was a small lump in the right side of the neck, about the size of a hazel nut. This was considered to be an enlarged jugulo-digastric gland, probably tuberculous, and operation was advised.

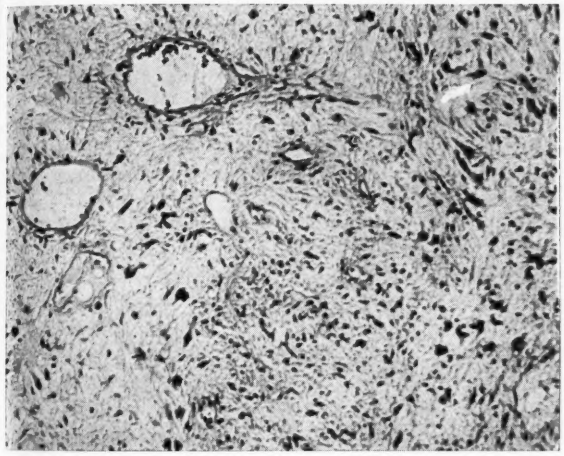

Fig. 164.—Section of tumour showing dilated blood-vessels and scattered cells in an œdematous stroma. Van Gieson. (× 85.)

Clinical examination revealed no other abnormality. Radiography of the chest was clear, and the erythrocyte sedimentation rate was normal.

At operation a mass, which was fairly firm and well circumscribed, shelled out unusually easily from behind the carotid sheath and there were no signs of other enlarged glands.

After operation the patient complained of inability to see clearly and of headache after trying to use her eyes.

On examination there was a notable degree of enophthalmos, ptosis, and myosis on the right side. Dryness of the skin was not evident. It was thought at first that the cervical sympathetic chain had been damaged at operation, but the persistence of the Horner's syndrome and the pathological report suggested another explanation. The pathological report read as follows :—

" Sections of the tumour show no evidence of lymphoid tissue. It consists of an encapsulated mass of connective tissue which is œdematous and contains multiple microcysts and many widely dilated capillaries and blood spaces with areas of hæmorrhage. In the capsule at one point is a small nerve-bundle. Scattered throughout the stroma are many irregular cells with pale vesicular nuclei, mostly rather elongated and occurring in groups and occasionally forming parallel bundles with some degree of regimentation in places. The connective tissue stains orange-brown with van Gieson's stain, with occasional patches of more typical collagen staining pink. Silver impregnation does not show any nerve elements.

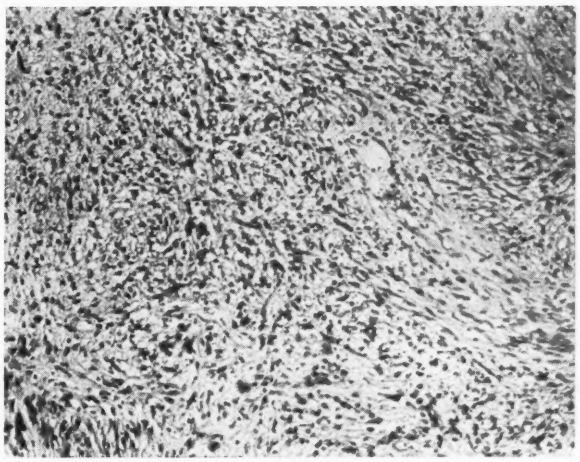

Fig. 165.—A more cellular part of the tumour, showing some bundle formation. Van Gieson. (× 85.)

" The appearances are those of a *neurilemmoma* (neurinoma) of mixed type, mostly type B. There is no evidence of malignancy."

Subsequent Progress.—The Horner's syndrome persisted to a marked degree for about two months. When

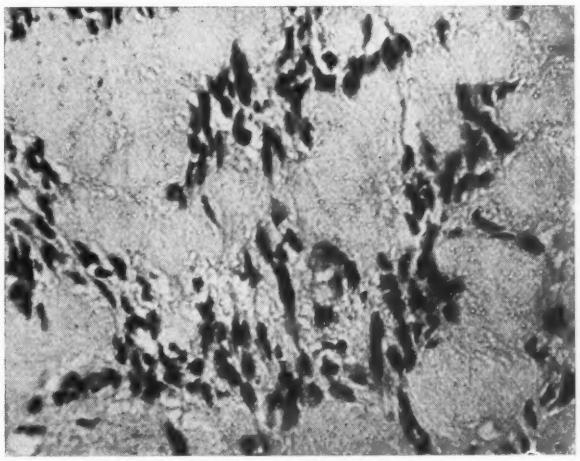

Fig. 166.—Part of tumour showing irregular regimentation of nuclei. Van Gieson. (× 375.)

last seen, six months since operation, it was only noticeable on careful inspection, and the patient was in excellent health.

It is an attractive theory that the tumour arose from a ramus of the cervical sympathetic chain.

I am indebted to Drs. Hamilton and Ross for the pathological report.

ENDOMETRIOMA OF THE CÆCUM CAUSING MUCOCELE OF THE APPENDIX

By PHILIP SHEMILT

SURGICAL REGISTRAR, ST. THOMAS'S HOSPITAL

MUCOCELE of the appendix is an uncommon condition. In this report an unusual cause of it is described in which an endometrioma of the cæcum was found to be obstructing the lumen of the appendix. No record could be traced of this occurrence.

In 1876 Frere first described a mucocele of the appendix, and Jones and Carmody collected 400 cases from the literature up to 1936. It is found in approximately 0·24 per cent of all appendicectomies (Mayo and Fauster, 1932). Four cases in 1850 appendicectomies have been recorded at St. Thomas's Hospital in the past ten years.

The comparative rarity of the condition is accounted for by the unusual combination of circumstances which determine its onset (Grodinsky and Rubnitz, 1941), and which are :—

1. Localized obstruction to the lumen of the appendix.
2. Preservation of its blood-supply.
3. Excess of secretion of mucin over absorption within a clean but not necessarily sterile lumen.

The disease is one of middle life and appears to be commonest in females. Usually the only symptom is pain of a mild colicky nature in the right iliac fossa which is rarely severe. Perforation can occur, but, when it does, does not give rise to any characteristic signs and symptoms. Often the patient gives a history of several attacks of mild pain lasting a few hours at intervals of several months. Nausea and vomiting are uncommon, there is usually no pyrexia, and unless the rare complications of intussusception or intestinal obstruction occur (Martin, 1934; Fraser, 1937) there is no alteration of bowel habit.

It is not surprising that the condition is rarely diagnosed before operation, because the physical signs are not pathognomonic. The commonest finding is tenderness on palpation in the right iliac fossa, associated with some guarding of the right lower rectus muscle. Occasionally a swelling may be felt. Although a filling defect of the cæcum may be outlined after barium enema X-ray examination, there is usually no radiological abnormality. Preoperatively the diagnosis of appendicitis is usually made, but sometimes it is mistaken for a carcinoma of the cæcum.

The treatment is appendicectomy. The appearance of the mucocele varies. The appendix is distended, appearing globular, conical, or egg-shaped, depending upon the site of the obstruction. The wall may be greatly thinned and almost transparent, sometimes showing diverticula or even a perforation, through which exudes the typical jelly-like mucin.

In early cases microscopical examination of the wall reveals hyperplasia of the mucous membrane, with irregular papillary formation of the secreting columnar cells. At this stage the muscle coat is hypertrophied, with round-cell infiltration of the muscularis mucosa.

As distension increases the wall of the appendix becomes atrophied, with disappearance of lymphoid tissue and mucous membrane and with increased submucous fibrous tissue (Phemister, 1915). The contents vary from a small amount of mucinous tacky fluid found in early cases to the gelatinous opalescent contents of the fully formed mucocele.

The rate of the progress of the condition is unknown, although from the history it is likely that the mucocele is gradually formed over many months or years. However, Wells (1937) found that obstruction of the rabbit's appendix by ligature and with preservation of the blood-supply could result in the formation of mucocele in one to two months.

In recurrent acute appendicitis, as Williams and Boggon (1934) have pointed out, a fibrous stricture is present in the submucosa. It is quite possible that this type of obstruction is the commonest cause of mucocele.

In the case recorded below it was found that endometriosis affecting the cæcum near the base of the appendix had by its fibrous-tissue reaction caused blockage of the lumen. From the size of the implant ir was probable that this had been present for some months. Added interest was given when it was discovered that the endometrioma contained a few glandular elements characteristic of the cervix of the uterus, which is a most unusual finding.

CASE HISTORY

HISTORY.—The patient was an unmarried woman of 47, who was admitted as an emergency to St. Thomas's Hospital on March 18, 1948, complaining of abdominal pain of thirty-six hours' duration. Eight years before she had been admitted to the Rotunda Hospital, Dublin, on account of severe dysmenorrhœa, with pain starting one week before her periods for several years. On that occasion a chocolate cyst of the ovary was removed. No lasting improvement was obtained, and in 1946 curettage was performed—without relief. The dysmenorrhœa, with some excessive loss at her periods, continued. The last menstrual period was one week before admission. The patient had had no other serious illnesses.

The recent attack of pain began in the right subcostal region, later moving to the right iliac fossa and persisting as a dull ache made worse on movement. There was no nausea or vomiting, but there was loss of appetite from the onset of the pain. The bowels, which were usually somewhat constipated, had not moved for two days. Micturition was normal.

ON EXAMINATION.—The temperature and pulse were normal. The tongue was clean. The abdomen was not distended, but there was some tenderness without rigidity in the right iliac fossa. No masses could be found and the previous midline laparotomy scar was sound. Rectal and vaginal examinations were negative.

PROGRESS.—The patient's symptoms were mild, and as they improved with rest in bed the condition was observed for the next few days. However, when the patient was allowed up the pain returned and the

previously normal temperature rose to 99°. A diagnosis of mild acute appendicitis was made.

Operation was therefore advised and was carried out on March 24 by Mr. Mitchiner. At laparotomy the appendix was found to be greatly distended, measuring

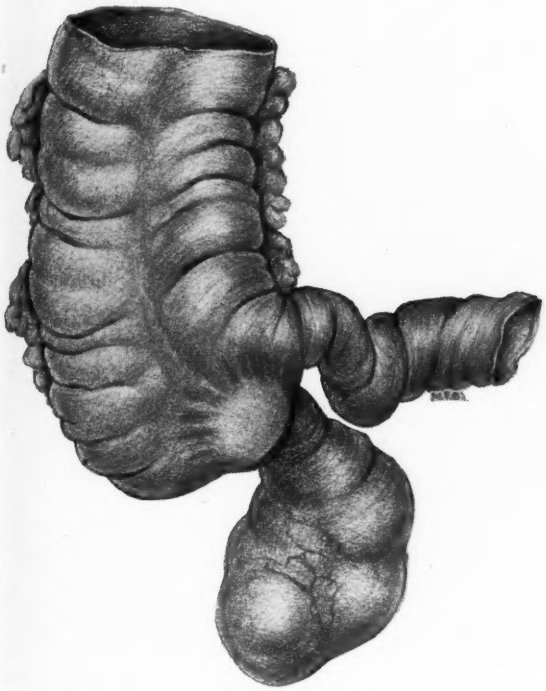

FIG. 167.—Mucocele, showing the site of the endometrioma in the wall of the cæcum.

5 cm. long and 3·5 cm. in its broadest diameter. It formed a typical mucocele of a yellowish-pink colour and in several places the wall was thin and translucent although no perforation had taken place. (*Figs.* 167, 168.)

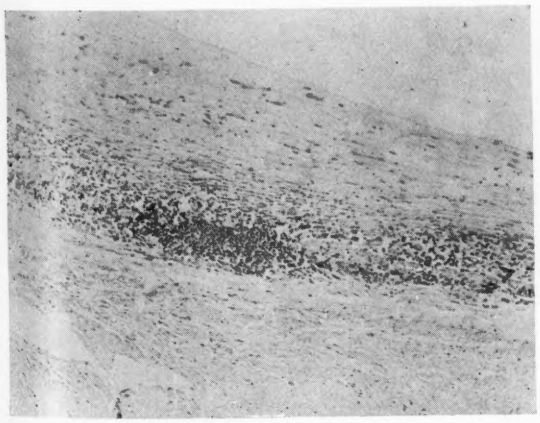

FIG. 169.—Photomicrograph of section of the wall of the mucocele. Disappearance of mucous membrane and degeneration of the muscle coat. (× 45.)

In the anterior wall of the cæcum at the base of the appendix a firm plaque of tissue could be felt. It measured about 1·5 cm. in diameter and was causing obstruction to the lumen of the appendix. It appeared to be composed of fibrous tissue, and later microscopical examination showed that the fibrous tissue reaction was due to an endometrioma.

Removal of the tumour and appendicectomy was performed by resection of the affected part of the cæcum, the ileocæcal valve, and the terminal 2 in. of ileum, which were bound down by fibrous tissue. Bowel continuity was restored by anastomosis of the ileum to the divided end of the cæcum. Following the operation the patient made an uninterrupted recovery and

FIG. 168.—Mucocele of the appendix.

was discharged from hospital fourteen days after the operation. Since then she has remained well. An artificial menopause by radiation was advised for the treatment of the endometriosis.

HISTOLOGICAL APPEARANCES (*Figs.* 169, 170).—Histological examination of the cæcal tumour shows that the

FIG. 170.—The endometrioma of the wall of the cæcum. Above is an islet of endometrial tissue and below this a racemose gland, characteristic of the cervix.

appearances are those of an endometrioma. Islets of varying sizes composed of endometrial glands with surrounding stroma are present throughout the bowel wall. Most of the glands are typical of those of the body of the uterus, but there are a few which are characteristic of the cervix, being racemose and composed of columnar cells with clear cytoplasm.

COMPLICATIONS OF MUCOCELE

In the case reported the wall of the appendix was intact and no leakage of its contents had taken place. Rupture is, however, the commonest complication and results in pseudomyxoma peritonei (Phemister, 1915). This was first described by Frankel in 1901 and has been reproduced experimentally (Grodinsky and Rubnitz, 1941).

Less commonly the appendicular swelling gives rise to an intussusception (Fraser, 1943).

Rarely acute inflammation occurs (Early Latimer, 1940), with exacerbation of abdominal pain and vomiting. The mucocele then contains blood-clot and mucous, with acute inflammatory changes in the wall.

Associated carcinoma of the appendix has been reported (Waugh and Findlay, 1937).

SUMMARY

The literature on mucocele of the appendix is reviewed. One case due to endometriosis of the cæcum is added. No record has been traced of this occurrence. The microscopical appearances of the area of endometriosis revealing the presence of cervical glands is shown.

I should like to thank Mr. P. H. Mitchiner, C.B., C.B.E., for his permission to publish this case and for his help in the preparation of this article. I am also greatly indebted to Dr. J. Bamforth for the photomicrographs and the description of the microscopical findings of the case; and to Miss Ball for her drawings of the specimen.

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CRUSHED CHEST SYNDROME WITH MEDIASTINAL EMPHYSEMA THE RESULT OF COMPRESSED AIR

BY W. M. TOONE

CASE REPORT

W. C., aged 55, was employed in a shipyard in North Vancouver. On Nov. 21, 1947, he was testing a boiler with compressed air when the boiler burst under the pressure and he was found lying in a pool of blood. He sustained a severe laceration of the left side of the neck, a rent that began at the sternal notch and ended behind the left ear. It divided the sternomastoid and exposed the carotid sheath. He was greatly shocked, but his chief complaint was severe pain in the chest. Shock therapy was instituted and after resuscitation

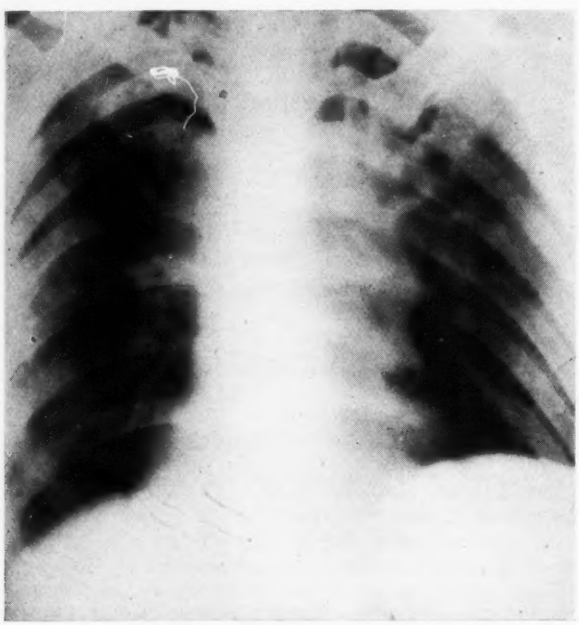

FIG. 171.—Radiograph showing widening of the mediastinal shadow above the heart. There is some mottled infiltration in both lung fields. Diagnosis of crushed chest syndrome with mediastinal emphysema and fracture of ribs.

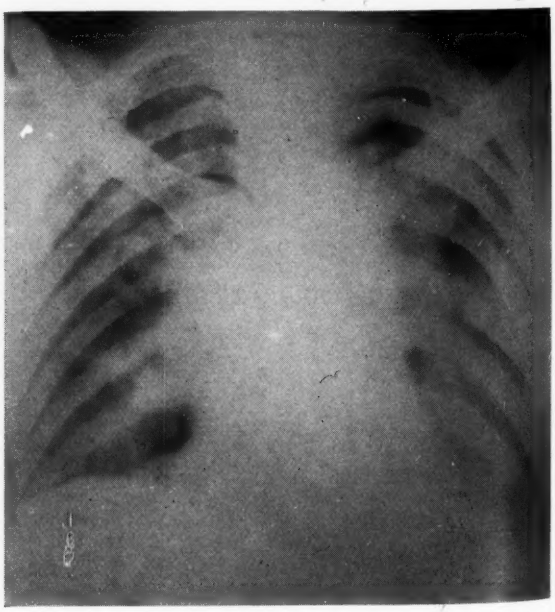

FIG. 172.—Radiograph taken later on Nov. 23 shows that the infiltration throughout both lung fields is somewhat suggestive of a pneumonia, although the appearance of interstitial and mediastinal emphysema is still present. The right lung root shows an increase in density.

his neck wound was dealt with, this being some eight hours after the injury and was done then because of the continual bleeding.

He continued to have pain in breathing and portable X-ray showed fracture of several ribs on the left side, five, six, seven, and eight at least (*Fig.* 171). Fine

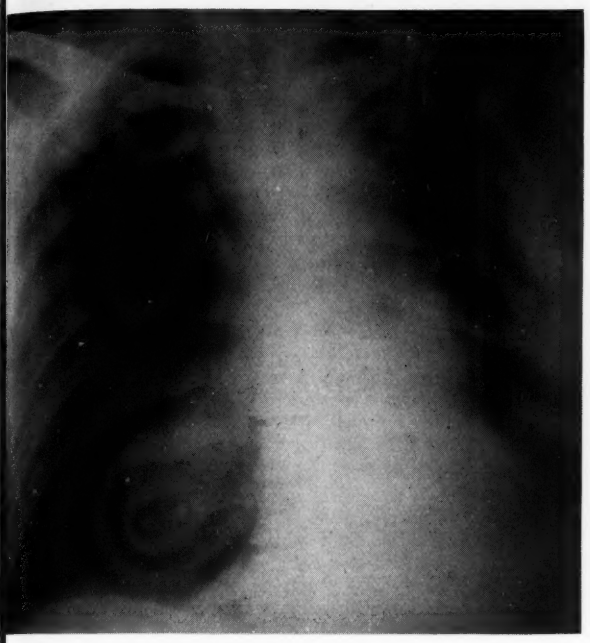

FIG. 173.—Radiograph showing a pneumothorax of the right side compressing all lobes. 600 c.c. of air was removed and the patient became comfortable.

crackling râles could be heard all over the chest but no subcutaneous emphysema was present. Cardiac and liver dullness was markedly diminished. The heart-sounds were distant but normal; no pericardial rub was heard, but there were numerous loud crackling sounds down both sides of the sternum, and some of these were synchronous with the heart-beat.

On the morning of Nov. 23 the patient became unconscious, the pulse very rapid and thready, his breathing shallow, rapid, and distressed, and he was markedly cyanosed in the face and extremities. It was decided that his condition was due to tamponade of the great veins from the pressure of air in the mediastinum. A needle was accordingly inserted into the retrosternal space at the level of the second interspace. Some 150 c.c. of free air was withdrawn, which was under sufficient pressure to push out the piston of a syringe, though the actual pressure was not measured. Following this procedure he dramatically improved, the pulse steadied and became stronger, and his colour gradually returned. How near he had been to death was subsequently illustrated, for areas of skin that had sustained pressure, his heels, buttocks, and shoulders, turned black and sloughed.

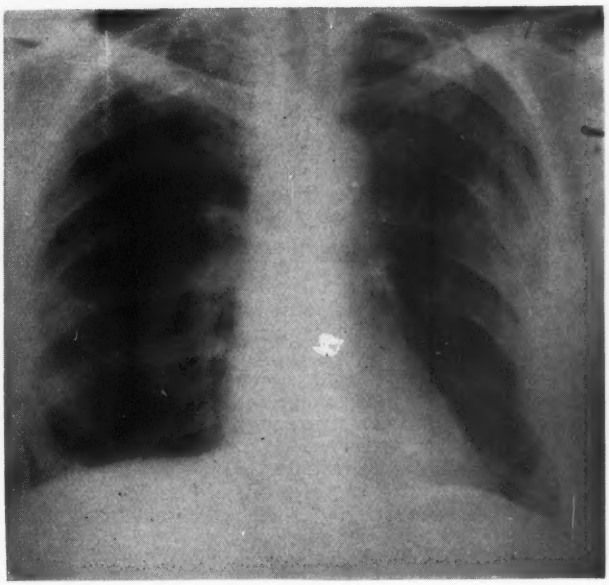

FIG. 174.—Film taken on Dec. 27, 1947, showing the mediastinal shadow back to normal and the lung fields clearing satisfactorily.

Patient's temperature became elevated and he began to cough up dark, bloody sputum. A bronchopneumonia had developed, probably due to blast effect. Penicillin and sulpha therapy had, of course, been instituted from the start in anticipation of this event.

On Nov. 27, late in the evening, the patient suddenly developed a marked dyspnœa. A shift of the mediastinum to the left was noted and a tympanitic note to percussion over the right chest.

An uneventful recovery over a prolonged period of time took place (*Fig.* 174). Mentally the patient showed some confusion at times but this gradually cleared, although he retained no memory of the accident. He returned to work March 1, 1948.

Comment.—The mechanism of this man's injury was presumably blast effect from the burst boiler. A mediastinal emphysema occurred, which was relieved by aspiration of air under pressure by inserting a needle in the right second interspace.

REVIEWS AND NOTICES OF BOOKS

Tuberculosis of the Knee-joint. By JOHANNES MORTENS. $6\frac{5}{8} \times 9\frac{7}{8}$ in. Pp. 550 + xvi, with a number of illustrations. 1949. Copenhagen: Einar Munksgaard. (London: H. K. Lewis & Co.) Dan. kr. 30.

THIS monograph is based upon an exhaustive study of the literature and an analysis of 181 cases of tuberculosis of the knee treated at the Coastal Hospital, Refnaes, and the Finsen Institute, Copenhagen. The object of the work has been to ascertain, as far as possible, whether treatment should follow exclusively conservative lines during early childhood or if treatment by excision at an early age as practised in America is justifiable. The author concludes that it is quite unjustifiable to treat all cases of tuberculosis of the knee in childhood by excision irrespective of the age of the patient or the extent of the disease. He recommends that all cases should be submitted to conservative treatment in the first instance, and his investigations have shown that when the disease is synovial in type without destruction of the articular surfaces, conservative treatment alone will result in healing with a functional result which far surpasses the results seen after excision. He believes that excision should never be performed before the age of 9 years, and not before 13 or 14 when X-ray examination shows signs of involvement around the epiphysial lines, if the risk of post-operative shortening is to be avoided. The primary indication for excision is considerable destruction of the articular surfaces, but resection may be considered during later childhood in cases of synovial disease which have not become quiescent after conservative treatment extending over a period of six to seven years. Treatment of the disease in adults is not considered at any length, but excision is regarded as the method of choice and conservative treatment employed only in incipient cases and mild cases in which the disease is localized entirely to the synovial membrane. The views expressed do not differ greatly from those which are held in Great Britain at the present time.

The English translation by Axel Andersen is excellent. A comprehensive bibliography containing some 548 references and the clinical records of 150 cases are appended.

British Medical Association. Proceedings of the Annual Meeting, 1948. $6 \times 9\frac{3}{4}$ in. Pp. 415. 1949. London: Butterworth & Co. (Publishers) Ltd. 35s.

THE British Medical Association is to be congratulated on bringing out such an interesting volume containing all the papers and discussions that took place at Cambridge during the Annual Meeting in 1948. This is a new departure, but, one which we hope will be continued, for the volume is a mine of information. Messrs. Butterworth have done justice to the material they had to publish, for the different papers are set out in a lucid style with which we have now become familiar, and can be recognized as the Butterworth setting and format.

Sir Lionel Whitby's Presidential Address on the changing face of medicine, and Dr. Whittle's article on medicine at Cambridge make interesting reading to every medical man.

Although every section of medicine was very adequately covered, the surgical sections are of most interest to the readers of the JOURNAL.

In the section on surgery Denis Browne dealt with tuberculous adenitis in an exhaustive fashion and favoured as the best form of treatment incision into the glands and expression by force. This treatment was not advised by subsequent speakers.

Sir Cecil Wakeley's lecture on carcinoma of the breast was practical to a degree, and contained the results of thirty years' clinical experience as a surgeon.

Bronchial carcinoma is fully covered by R. C. Brock, G. A. Mason, R. R. Trail, and F. H. Young.

The treatment of lesions of the intervertebral disk is dealt with by Norman Capener, N. M. Dott, and others.

Perhaps the most fascinating paper in the volume is that by Wood Jones, who deals with the present position of primate anatomy; a fascinating and illuminating paper, which should be read by every anatomist and every medical man and woman.

Surgical Technique and Principles of Operative Surgery. By A. V. PARTIPILO, M.D., F.A.C.S., Associate Clinical Professor of Surgery, the Stritch School of Medicine of Loyola University; etc. With a Foreword by ALTON OSCHSNER, M.D., F.A.C.S. Fourth edition. $7\frac{1}{8} \times 9\frac{7}{8}$ in. Pp. 676, with 547 illustrations. 1949. London: Henry Kimpton. 75s.

THE fourth edition of this large volume on surgical technique has been thoroughly revised by Professor Partipilo and his eight contributors. Throughout the book there is a distinct simplicity and clarity which is a rarity in medical and surgical books of to-day. First things come first, and there is an excellent chapter on general principles of operative surgery dealing with the real fundamentals of surgery. Other chapters are devoted to sutures, and knots and methods of tying them.

The whole subject of operative surgery is well and truly covered in a very practical way and the illustrations are on the whole very good, but a few are misleading, e.g., the drawing of cæcostomy depicts a glass rod under the cæcum, which is surely unnecessary.

The real criticism of the book is that a large number of unnecessary operations are depicted. Murphy's button is only to be found in medical museums in this country. Local excision of peptic ulcers is not a treatment which should be advocated to-day. It is not necessary to describe and illustrate eight different methods of performing gastrostomy. The operation is rarely performed to-day, and several of the methods described could not be performed on a small and contracted stomach such as one sees in cases of carcinoma of the œsophagus.

However, these criticisms do not outweigh the value of this book, because it is a comprehensive volume and it is full of sound surgical common sense and will prove of real value to the advanced student reading for higher surgical qualifications. It should also be valuable as a book of reference.

The 1948 Year Book of Urology. Edited by OSWALD S. LOWSLEY, M.D., F.A.C.S., Director, James Buchanan Brady Foundation, New York Hospital; etc. $4\frac{5}{8} \times 7$ in. Pp. 445, with 79 illustrations. 1949. Chicago: The Year Book Publishers Inc. (London: H. K. Lewis & Co.). 26s.

THE numerous articles on urological subjects appearing in a single year in the general medical and surgical journals, apart from those in the special urological publications, form a symposium which every urologist would like to have readily available for reference. Their collection, whether by tearing out the relevant pages or by elaborate card indexing, involves much time and labour. In this volume the work has been done, and

they are presented in a handy form with an index. The successive annual volumes of the *Urological Year Book* are the milestones in the march of this rapidly advancing branch of surgery.

Injentos de Piel. By ALBERTO RAUL BEAUX, Professor Libre de Medicina operatoria de la Facultad de Ciencias médicas de la Universidad nacional de la Plata. 7 × 10⅜in. Pp. 257, with 138 illustrations. 1947. Buenos Aires: El Ateneo. Price not stated.

THIS monograph of 257 pages, written in Spanish, describes in detail all the recognized methods of free transplantation of skin. The earlier chapters are devoted to cross-grafting (homo- and hetero-grafts); the histological changes and the phases in the establishment of nutritional supply in grafted skin; the classification of grafts according to thickness, from the thin Ollier-Thiersch dermo-epidermic graft to the full-thickness Wolfe-Krause graft. Instructive photomicrographs are well reproduced and there are useful diagrams indicating the constituents of grafts of differing thickness.

Reasons are advanced for selecting one type of graft in preference to another in the treatment of a particular condition, and pre- and post-operative care are given the important consideration they deserve. In the section dealing with choice of donor area reference is made to the use of full-thickness skin from the supraclavicular region as recommended by Brown and Cannon.

A well-illustrated chapter describes all the mechanical aids to skin-graft cutting. The frequent appearance of drawings of the Humby knife in use suggests that this excellent instrument is as popular with the author as it is with many plastic surgeons in this country. There is nothing new in the chapter on methods of fixation for grafts and dressings. The repair processes which occur in the donor area are described in considerable detail and are illustrated by numerous photomicrographs.

Photographic case records, showing most gratifying results, come from the service of Dr. Malbec.

Twelve pages are devoted to the bibliography: this and the index of authors should prove extremely useful to those wishing to follow the literature of the subject. There is a good general index.

The author is to be congratulated on the production of a thoughtful and scientific thesis which will be of particular value to those who read Spanish: even to those who cannot read the text, its illustrations, diagrams, and bibliography will be of considerable interest.

Paravertebral Block in Diagnosis, Prognosis, and Therapy. Minor Sympathetic Surgery. By FELIX MANDL, M.D., F.I.C.S., Professor of Surgery, Hodassah University Hospital, Jerusalem. Translated by GERTRUDE KALLNER, M.D. With a Foreword by MAX THOREK, M.D., F.I.C.S., Chicago. 6⅛ × 9 in. Pp. 330 + xvii, with 20 illustrations. 1948. London: William Heinemann (Medical Books) Ltd. 32s. net.

THIS is an excellent book—well translated. It is clear, concise, honest, and comprehensive, and brings into a relatively small scope a store of knowledge which is obviously the result of the author's long experience and wide reading. Dr. Mandl did in fact lose in Vienna in 1939 the painstaking records of much pioneer work on the sympathetic nervous system carried out over a period of almost twenty years, and the fact that he has during the last ten years been able to repeat much of it and record the results of his findings and his thoughts in such a succinct account of the subject as this book, speaks volumes both for his industry and his inherent belief in the vocation of medicine. There is a delightful philosophy underlying the book founded on the thesis that pain is a fundamental factor in human life and yet the handling of the subject, its diagnosis, prognosis, and therapy is eminently practical, even if here and there already a little out of date. The use of alcohol injections is rightly rather frowned upon, but phenol injections—now well established—are discussed as rather an experimental measure. The chapter on technique is very good and of the many diseases in which paravertebral block may be employed, the description of angina pectoris stands out. It is noteworthy throughout the book that the failures of the method receive just as fair treatment as do the successes. Illustrations are few, but such as are used are clear and amply illustrative of the procedure described in the text. The book would be an undoubted asset to the library of the physician, the neurologist, the surgeon, and the anæsthetist alike.

Cunningham's Manual of Practical Anatomy. Revised and edited by JAMES COUPER BRASH, M.C., M.A., M.D., F.R.C.S. (Edin.), F.R.S.E., Professor of Anatomy, University of Edinburgh. Eleventh edition. 4¾ × 7¼ in. Volume I: General Introduction; Upper Limb; Lower Limb. Pp. 387 + xix, with 191 illustrations, many coloured (44 as plates). 1949. London: Oxford University Press 21s.

IT is some sixty years since the late Professor D. C. Cunningham published his *Manual of Practical Anatomy*, and since that date many thousands of medical men the world over have used the well-known book in its various editions. It is a byword in the medical profession. The new eleventh edition retains the same standard of approach to dissection, and several new drawings and radiographs have been added.

The General Medical Council, in its wisdom, has still insisted that the medical student must dissect the whole body, and, this being the case, Cunningham's manual still holds the field as the best book on this important subject.

No student of medicine can afford to be without this book, and the post-graduate and the teacher of anatomy will find it invaluable. Every surgeon should have it as a reference volume.

Medical Photography. Radiographic and Clinical. By T. A. LONGMORE, Hon. F.S.R., Principal, Kodak School of Medical Radiography and Clinical Photography; etc. With a Foreword by Brigadier D. B. MCGRIGOR, O.B.E., M.B., Ch.B., D.M.R.E., Hon. F.S.R., Consulting Radiologist, The War Office; President, British Institute of Radiology, 1939–42. 4¾ × 7¼ in. Pp. 1008, with 320 illustrations and 120 plates. 1949. London: Focal Press. 50s.

THE medical profession of to-day fully realize the importance of photography in all branches of medicine. Photographic records are easily kept and are worth many pages of clinical notes. It is not five years ago since Mr. Longmore published his book on medical photography and now the fourth edition makes its appearance. During the interval the book has grown to double its original size and to-day conrains over a thousand pages. It is to be hoped that the author will see his way in the future to divide the book into two, one being devoted to radiography and the other to medical photography. There can be no doubt that the author has performed a signal service to the medical world in gathering together such a massive collection of relevant facts and figures into a comprehensive volume. It will prove to be a unique book of reference to the student, the amateur, and the professional photographer.

Hospitals to-day may be said to be "photographically minded" and realize that a department devoted to this subject is just as important as the X-ray department. It is to be hoped that before long every hospital in the

country will have a well-equipped photographic department.

This book deals with every technique in photography and the theory is excellent.

The illustrations are uniformly good, but the paper might have been better.

There is a useful and comprehensive formulary and glossary and a valuable " Index of Tables " which will prove of real value to those interested in medical photography.

An Introduction to Surgery. By RUTHERFORD MORISON, M.D., F.R.C.S., M.A., D.C.L., LL.D. and CHARLES F. M. SAINT, C.B.E., M.D., M.S., F.R.C.S., F.R.A.C.S. Fourth edition. $8\frac{5}{8} \times 5\frac{5}{8}$ in. Pp. 330 + xii, with 304 illustrations. 1948. Bristol: John Wright & Sons Ltd. 42s. net.

> " Some one or nobody knows I wot
> Who or which or why or what."
> —*Edward Lear.*
>
> " I have six trusty serving men
> Who taught me all I know,
> Their names are where and why and when
> and what and how and who."

RUTHERFORD MORISON died on Jan. 9, 1939, aged 85 years. He retired from the Chair of Surgery in Durham University, the Medical School of which is situated at Newcastle-on-Tyne, at the age of 61. He published his book, *An Introduction to Surgery*, in 1910. He himself regarded this book as his most valuable work and spent a great deal of time during his eighty-third year in preparing a new edition in collaboration with his old pupil, Professor F. M. Saint, of Capetown. (" Rutherford Morison and His Achievement ", the first Rutherford Morison lecture delivered by Professor G. Grey Turner at Newcastle-on-Tyne Infirmary, May 29, 1947.)

In the Preface to the third edition, published in 1935, Rutherford Morison stated, " The object of this book, as described in the first edition, is to aid the student in thinking out for himself the problems presented to him in the wards and in his text-books. The achievement of this object is attempted through the consideration of *general principles* and their application, by which it is realized that all the important pathological subjects to be studied are part of an all-embracing whole."

In this, the fourth edition, Professor Saint, one of his disciples, maintains this ideal: as he states, " Principles do not change though more may be discovered ". This edition has been published as requests continue to be received for the book, which has been out of print.

With the growth of surgical specialities, it is now more important than ever that those training to practise them, under the new health service, should have it impressed upon them from the start that the various branches of medicine and surgery are not independent but interdependent, and that the entrance into all these narrow modern specialities can only be through the portals of general medicine, surgery, and pathology. One can only repeat the first sentences of this book, in which it states that its object is to teach students to think and not to memorize, " Study without thought (memorizing) is futile and unproductive of understanding, whereas thought without study (irresponsible thinking) is not only unproductive but is often actually dangerous. Study with thought alone is productive and useful and leads to understanding. Knowledge without understanding is superficial . . . and cannot be applied, whereas knowledge with understanding gives maximal returns, as it can be readily applied and results in good judgement and wisdom ".

Every teacher and examiner will agree with these ideals. The first chapter is devoted to an explanation of the words, terms, and language of surgery, the definition of inflammation and the results of the reaction of the body to injury by whatever cause it may have occurred.

Then the nature and results of wounds are considered, and also the fact, which was realized early in his career by the author, that the treatment of a wound wherever it occurs is of fundamental importance and the ideal dressing for a wound is *skin*.

The chapter is what one would expect from one of Lister's special clerks, which post he occupied in 1873. He told Professor G. Grey Turner that " he considered Lister the greatest man he had even known and spoke of him as an inspired genius ". All through his life he was attracted to antiseptic surgery, the outcome of which was the invention and use of B.I.P.P. to combat the sepsis in the wounds of the 1914–18 war.

Then follow in orderly sequence the signs, symptoms, and complications of acute inflammation due to infection and the result of specific infectious diseases. The pathology of the various conditions is fully stressed, and explained with numerous excellent illustrations, and a few typical cases are quoted from the author's immense experience to impress some special aspect on the mind of the student. He particularly quotes his own errors in diagnosis when discussing the chronic inflammatory conditions of syphilis and tuberculosis, especially the difficulty of distinguishing them in certain cases from neoplastic disease. The dire effect of superadded sepsis in these conditions is graphically portrayed. Certain statements made, rather dogmatically, would not be accepted wholeheartedly to-day, e.g., " syphilis and tubercle is a deadly combination "; " persons who have recovered from tubercle in their youth are exceptionally liable to cancer in their later years "; " sarcoma is not infrequently related to a single recent severe injury ". The author is convinced that injury is an aetiological factor in the production of malignant disease. This would be agreed by most surgeons, the experimental evidence alone is overwhelming, but it is usually a slight injury perhaps often repeated. A sarcoma never follows a simple fracture nor does osteomyelitis attack the fractured bone.

The operation described by the author, which took place more than fifty years ago for a psoas abscess, would not be practised nowadays, but, on the other hand, the pendulum is perhaps swinging too far in the opposite direction, and if surgery is practised by open operation, then it must be as thorough in excision and removal of the infected tissue as it would be for malignant disease. He does, however, condemn indiscriminate amputation for sarcoma of bone (p. 205) and inclines to local operations and the use of Coley's fluid. The latter was never greatly favoured in this country, and is now rarely used, though it might be remembered with advantage to the patient.

The chapter on " The Serous Cavities " is very refreshing, particularly the section devoted to " some functions of the omentum ". It was in 1896 that he first did the operation of omentopexy known as the Morison-Talma operation for alcoholic cirrhosis, and demonstrated, at any rate, one use for this structure.

A chapter is devoted to " Indications for Operation " in which the principles of surgical treatment are duly stressed, viz.: (1) Remove the cause; (2) Combat the destructive effects of the lesion; (3) Assist the natural reaction; (4) Prevent complications. These principles of treatment will remain when time shall be no more.

Most surgical procedures except those necessitated by trauma are illogical; effects are treated and causes remain untouched. The logical aim of treatment is to remove the cause and thus achieve prevention, which is more than is included in the fashionable schemes of " positive health ".

As is pointed out, the cause could be removed more often if the underlying pathology was studied, with the assistance of X rays and the various scopes, and all traumatic wounds were explored to ascertain the extent of such injury.

The book finishes appropriately enough with a series of pathological illustrations to which the foregoing principles can be applied.

It has obviously been difficult for Professor Saint to carry out his task as editor and still keep the book essentially as Professor Rutherford Morison wrote it. On the whole he has succeeded, but it might have been better to have described modern methods of blood transfusion and laid more stress on modern chemotherapy.

The publishers are to be congratulated on the number and excellence of the illustrations, especially those portraying the various lesions produced by syphilis. These lesions are not seen in the out-patient departments nowadays to anything like the extent they were met with formerly.

The student consequently has a very vague idea of the importance of always remembering the possibility of syphilis.

The first Rutherford Morison Lecture was delivered at the Victoria Hospital, Newcastle-on-Tyne, on May 29, 1947, by Professor G. Grey Turner, who was one of his dressers, his house surgeon in 1899, and his private assistant for nearly fifteen years. He chose as his title "Rutherford Morison and His Achievement".

This lecture, together with his book, really describes a definite epoch in the development of surgery in this country. They should be bound together.

The book is more than is claimed for it by its title. As an introduction to surgery it can with safety be put into the hands of the junior clinical student, but he should read it again before his final examination and many times during his post-graduate surgical career as he gradually realizes surgical diagnosis is much more than a guessing competition. He would then realize the surgical truth expressed on p. 259: "True advance in surgery is, to a certainty, not to be expected in the direction of bolder operating, but through increased knowledge of aetiology, pathology, diagnosis, and prognosis, so that the earliest manifestations of disease may be recognized in time to prevent the serious happenings so frequently dealt with now."

Diagnostic Roentgenology. Edited by ROSS GOLDEN, M.D., Professor of Radiology, The College of Physicians and Surgeons, Columbia University; Doctor of Radiologic Service, The Presbyterian Hospital, New York. Renewals for Vol. I, II. Edinburgh: Thomas Nelson & Sons.

THIS is a loose-leaf book and is the third issue of renewals for *Diagnostic Roentgenology* since its original publication.

It is six years since the last set of new pages was sent to the purchasers of the original book.

The publishers have already published in the past a loose-leaf system of medicine and one of surgery.

There are twenty-two contributors to these two volumes. The articles are therefore somewhat unequal in style and content. They are, however, all by recognized authorities in their specialty.

The numbering of the pages is a little confusing at first, but would give no difficulty to one who has the original book and the second set of loose leaves published in 1942.

In the introduction the editor rightly stresses that the value and importance of an X-ray examination depends on the experience and skill of the radiologist.

The various regions are considered in order from a diagnostic and radiological point of view, beginning with paranasal sinuses and the mastoids. Then the chest is considered and then the alimentary canal. The radiology of the chest is confined to that of the lung; the heart will have to be considered in a further publication when the methods get more standardized.

The monographs on the digestive tract and genito-urinary system are models of what such articles should be.

The volumes end with an article on uterotubography. The word is a new one, but it explains what is meant.

The illustrations are profuse and excellently produced. It is a book which will be almost necessary in any diagnostic X-ray clinic.

Neuroradiology. By ALEXANDER ORLEY, M.D., D.M.R.E., Consulting Radiologist, West End Hospital for Nervous Diseases. Pp. 421 + xii. 1949. Oxford: Blackwell Scientific Publications Ltd. 63s.

THIS book in some respects might be considered as a second edition of Wakeley and Orley's original book on the subject which was published in 1938. The present volume only has the name of Orley on the title page while several of the illustrations are the same as appeared in the earlier book.

The Second World War brought an enormous influx of cranial injuries to the neurological surgeons of this country and as a result of this the treatment of head wounds has been considerably altered. Improvements in X-ray technique have been of great assistance to the surgeon.

This book covers the whole field of neurology and its relation to radiology. It thus covers a field that has not been covered before and should prove of great worth to the neurologist, neurosurgeon, and pathologist. As a work of reference it will be used by all clinicians who have neurological problems to solve.

The book is beautifully produced on good paper, the text is lucid and clear, while the illustrations are excellent and are quite a feature of the volume. The bibliography is very comprehensive but it could have been more up to date.

Ernia del Disco e Sciatica Vertebrale. By FRANCESCO DELITALA and AUGUSTE BONOLA. 8vo. Pp. 213, with 108 illustrations. Bologna: Cappelli. L. 1800.

ANY monograph issuing from the Rizzoli Institute at Bologna is sure of a respectful reception, and this one on hernia of vertebral disks and sciatica of that origin is well worth study. It stems from an experience of 217 cases operated upon between 1934 and 1938, and reflects the developments of technique in the course of that time common to other clinics, though the authors deplore the frustrations and delays to progress caused by the war years.

The subject is well covered. It begins with the history of the syndrome, the anatomy, physiology, aetiology, and pathogenesis. There is then a record of some interesting experimental work on production of the lesion in the cadaver, followed by chapters on the histopathology, clinical picture, diagnostic methods, and operative treatment.

Finally, there are case histories of 21 patients, with photographic records of the operative findings and explanatory diagrams. The whole book is well illustrated. The authors' attitude to the problem is one of moderate enthusiasm and prudent caution. A book well worth reading. There is an extensive bibliography.

Neoplasms of Bone and Related Conditions. Their Etiology, Pathogenesis, Diagnosis, and Treatment. By BRADLEY L. COLEY, M.D., Attending Surgeon, Bone Tumor Department, Memorial Hospital for Cancer and Allied Diseases; Assistant Professor of Clinical Surgery, Cornell. $6\frac{7}{8} \times 10$ in. Pp. 765 + xiv, with 389 illustrations. 1949. New York: Paul B. Hoeber Inc. $17.50.

IT is right and proper that the son of the late Professor William Coley should write a book on tumours of bone,

as he must have had ample opportunity of seeing his father's cases.

This large volume deals with tumours of bone as seen and treated by a surgeon who is attached to the Bone Tumour Department of a large hospital. With the continued specialization in medicine and surgery to-day, such a book as this will be of value to the oncologist, the orthopædic surgeon, the general surgeon, the radiologist, and the general practitioner.

In this book the clinical and radiological aspects of neoplasms of bone are stressed, and little attention is paid to the microscopical pathology.

The classification of bone tumours is that used by the Bone Sarcoma Registry of the American College of Surgeons and is a very comprehensive yet very practical one.

The various types of bone tumour are well described and illustrated, and an adequate account of the treatment to be used is given in the various chapters.

An excellent chapter devoted to the effect of radiation on bone tumours is included; the use of mixed toxins is not advocated strongly.

The book will fulfil the purpose for which it was written, but the bibliography is almost entirely American and the name of Fairbank is not mentioned once, which surely is a grave omission.

Campbell's Operative Orthopedics. Edited by J. S. SPEED, M.D., and HUGH SMITH, M.D. Second edition. Two volumes. 1949. London: Henry Kimpton. £7 10s. net.

ORTHOPÆDIC surgery received a great stimulus from the two world wars. It was not till after the termination of the first world war that orthopædics became a special branch of surgery, yet in just over thirty years this branch has flourished and progressed more than any other, and to-day it forms one of the largest and most comprehensive sections of surgery.

Little wonder, then, that two large volumes containing some 1643 pages and 1141 illustrations can come from the Campbell Clinic and Faculty of the University of Tennessee. Living as we do in a mechanized age, traumatic surgery has come to the fore and the surgery of injury has an important place in the economic life of any nation.

The two volumes which make up this comprehensive and important treatise are the work of the members of the staff of the Campbell Clinic and the whole has been ably edited by J. S. Speed and Hugh Smith.

Every aspect of orthopædic surgery is covered and the rare conditions and diseases are described in detail and well illustrated. The more common orthopædic conditions receive scant attention. As reference volumes, this work could not be bettered, and resident medical officers and senior post-graduate students will find all they require to know for the higher surgical examinations within the pages of these volumes.

The illustrations are quite a feature of this work; many of them are original and they do enhance the value of the books and are a great help to the reader.

Observations on the Pathology of Hydrocephalus. By DOROTHY S. RUSSELL, Sc.D., M.D., F.R.C.P. Pp. 138 + vi. 1949. Issued by the Medical Research Council. London: H.M.S.O. 6s.

THE time is ripe for a clear, concise, comprehensive monograph on hydrocephalus, and Professor Dorothy Russell has produced just such a book. The monograph is in reality No. 265 of the special report series of the Medical Research Council. It is a very thorough survey of the morbid anatomy of hydrocephalus illustrated with excellent clear photographs and microphotographs.

It is interesting to note that Professor Russell does not make use of the word 'idiopathetic' in connexion with the origin of hydrocephalus, believing that such cases are in reality post-meningitic in origin.

This monograph will take its place as a book of reference on this important subject.

Surgery of the Hand. By STERLING BUNNELL, M.D., Honorary Member of American Academy of Orthopædic Surgeons. Second edition. Pp. 930 + xix. 1949. London: J. B. Lippincott Company. £5.

WE are glad to see the second edition of this excellent and comprehensive treatise on the surgery of the hand. Dr. Bunnell has had the good fortune of holding the position of Consultant in hand surgery to the Surgeon-General of the Army; an exceptional post, unique and unparalleled in the whole world. No other surgeon has had the opportunity of seeing the reconstruction of some 20,000 hands damaged in the late war.

The second edition of this lavishly illustrated book contains a wealth of clinical material which cannot be found in any other volume. Some of the coloured plates are beautiful examples of the blockmakers' art, and are of real value to the reader.

Every aspect of the human hand is considered, from the phylogeny and comparative anatomy to the injuries and tumours which may in themselves require amputation for their cure.

This volume is a classic in surgical literature; it should be read from cover to cover by every surgeon who has to treat cases of injury of the upper extremity. As a reference book it could not be bettered, the references are up to date and comprehensive, and of real value.

Neurosurgical Pathology. By I. MARK SCHEINKER, M.D., Assistant Professor of Neuropathology and of Medicine (Pathology), University of Cincinnati College of Medicine; etc. 6 × 9 in. Pp. 370 + xviii, with 238 illustrations. 1949. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 45s.

NEUROSURGERY, a comparatively new branch of surgery, has progressed at a rapid rate during the last three decades. It received considerable stimulation in the two world wars and is now established as a definite speciality in every country. Neurosurgical pathology, on the other hand, as a special branch of pathology is not so well known owing to the fact that there are few pathologists who are keen to take up this type of specialization. Professor Scheinker, from his great experience in medicine and pathology, is able to present a new and dynamic approach to this subject and his book is of immense practical value not only to the neurosurgeon but to the pathologist and neurologist as well.

It might be thought that a book devoted to neurosurgical pathology would be likely to produce a complicated and exhaustive classification of brain tumours, but in point of fact Professor Scheinker has given the reader a simplified classification based on clinical and pathological grounds.

Here is a book on a difficult and perplexing subject written in a lucid and understandable style beautifully illustrated and carefully printed on good paper, with an adequate bibliography.

A really useful book and one which will be the reference volume on neurosurgical pathology for many years to come.

On the Contributions of Hugh Owen Thomas of Liverpool, Sir Robert Jones of Liverpool and London, and John Ridlon, M.D., of New York and Chicago to Modern Orthopedic Surgery. By H. WINNETT ORR, M.D., Chief Surgeon, Nebraska Orthopedic Hospital, Lincoln, Nebraska. With a Supplement on Ridlon and His Share in Moulding

Orthopedic Surgery by ARTHUR STEINDLER, M.D., Professor of Orthopedic Surgery, State University of Iowa Medical School, Iowa City, Iowa. $5\frac{1}{2} \times 8\frac{1}{4}$ in. Pp. 253 + xiv, with 22 illustrations. 1949. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 25s.

THE early history of any branch of surgery is of interest to the medical historian and to the medical public as a whole. This book is devoted to the three great pioneers of orthopædic surgery: Hugh Owen Thomas, Sir Robert Jones, and John Ridlon. There can be no doubt that each of these three men left their mark in the surgical world, and to-day the younger generation of surgeons are familiar with their names.

It is most pleasing, however, to have concise, accurate, and short biographies of these three surgeons and to see how the influence of Thomas of Liverpool moulded the surgery of Ridlon, who himself has been styled the Father of Orthopædic Surgery in the Middle West. Most of the illustrations in this book have been culled from other volumes, but this in itself does not detract from its usefulness in giving the reader a clear-cut picture of three eminent men.

The author, a distinguished orthopædic surgeon, is well versed in the practice of his speciality both in England and America, and therefore he speaks with authority and interest on the contributions of these men.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Das Krebsproblem. Einführung in die Allgemeine Geschwulstlehre. By K. H. BAUER, Professor of Surgery, University of Heildelburg. $6\frac{3}{4} \times 10$ in. Pp. 758, with 71 illustrations, some coloured. 1949. Berlin: Springer. DM 42.; geb. DM 45.60.

Clinical Roentgenology of the Digestive Tract. By MAURICE FELDMAN, M.D., Assistant Professor of Gastroenterology, University of Maryland; etc. Third edition. 6×9 in. Pp. 901 + x, with 641 illustrations. 1949. London: Baillière, Tindall & Cox. 44s.

Clinical Aspects and Treatment of Surgical Infections. By FRANK LAMONT MELENEY, M.D., F.A.C.S. Associate Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; etc. With a Foreword by ALLEN O. WHIPPLE, M.D. $6\frac{3}{8} \times 9\frac{5}{8}$ in. Pp. 840 + xiii, with 287 illustrations. 1949. Philadelphia and London: W. B. Saunders Co. 60s.

Plaster of Paris Technic. By EDWIN O. GECKELER, M.D., Professor of Orthopædic Surgery, and Chief of the Fracture Service, Hahnemann Medical College and Hospital, Philadelphia. 6×9 in. Pp. 220 + xiv, with 236 illustrations. 1948. London: Baillière, Tindall & Cox. 16s. 6d.

Fractures and Dislocations for Practitioners. By EDWIN O. GECKELER, M.D., Professor of Orthopædic Surgery, and Chief of the Fracture Service, Hahnemann Medical College and Hospital, Philadelphia. Fourth edition. 6×9 in. Pp. 371 + xii, with 344 illustrations. 1948. London: Baillière, Tindall & Cox. 27s. 6d.

Essentials of Orthopædics. By PHILIP WILES, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S., Hon. Orthopædic Surgeon, Middlesex Hospital and King Edward Memorial Hospital; etc. $6\frac{1}{4} \times 9\frac{3}{4}$ in. Pp. 486 + xv, with 365 illustrations and 7 coloured plates. 1949. London: J. & A. Churchill. 42s.

Blood Transfusion. Edited by GEOFFREY KEYNES, M.A., M.D., F.R.C.S., Emeritus Surgeon, St. Bartholomew's Hospital. With sections by H. F. BREWER, RICHARD ELLIS, R. I. N. GREAVES, GEOFFREY KEYNES, F. W. MILLS, R. BODLEY SCOTT, ANTHONY TILL and LIONEL WHITBY. $5\frac{5}{8} \times 8\frac{1}{2}$ in. Pp. 574 + xii, with 110 illustrations. 1949. Bristol: John Wright & Sons Ltd. 52s. 6d.

Atlas of Plastic Surgery. By MORTON I. BERSON, M.D., formerly Director of the Department of Plastic and Reconstructive Surgery, The Downtown Hospital and Pan-American Clinic, New York City. $7\frac{7}{8} \times 10\frac{3}{4}$ in. Pp. 304 + xii, fully illustrated. 1949. London: Wm. Heinemann (Medical Books) Ltd. 75s.

Clinical Methods in Surgery. By K. DAS, M.B. (Cal.), F.R.C.S. (Eng. and Edin.), Surgeon, R. G. Kar (Carmichael) Medical Hospitals and Medical College Hospitals, Government of Bengal. Second edition. $7 \times 9\frac{3}{4}$ in. Pp. 255 + viii, with 249 illustrations. 1949. Calcutta: City Book Co. (London: H. K. Lewis & Co.). 35s.

An Account of the Schools of Surgery, Royal College of Surgeons, Dublin, 1789–1948. By J. D. H. WIDDESS, M.A. (Dublin), L.R.C.P. and S.I. With a Foreword by WILLIAM DOOLIN. $6\frac{5}{8} \times 9\frac{3}{4}$ in. Pp. 107 + viii, with 16 plates. 1949. Edinburgh: E. & S. Livingstone Ltd. 17s. 6d.

A Companion in Surgical Studies. By IAN AIRD, Ch.M., F.R.C.S., Professor of Surgery in the University of London; Director of the Surgical Unit, Postgraduate Medical School of London. $7\frac{1}{4} \times 9\frac{5}{8}$ in. Pp. 1060 + viii. 1949. Edinburgh: E. & S. Livingstone Ltd. 63s.

Illustrations of Surgical Treatment. Instruments and Appliances. By ERIC L. FARQUHARSON, M.D., F.R.C.S. (Eng. and Edin.), Assistant Surgeon, Royal Infirmary, Edinburgh. With a Foreword by the late Sir JOHN FRASER, Bt. Third edition. $6 \times 9\frac{3}{4}$ in. Pp. 391 + xii, with 319 illustrations and 61 plates. 1949. Edinburgh: E. & S. Livingstone Ltd. 25s.

Manual for Laboratory Work in Mammalian Physiology. By FRED E. D'AMOUR and FRANK R. BLOOD. $11 \times 8\frac{1}{2}$ in. A collection of 50 experiments, fully illustrated. 1949. Chicago, Ill.: University of Chicago Press. (London: Cambridge University Press.) 15s.

Les Problèmes du Traitement des Ulcères perforés gastro-duodénaux. La Gastrectomie d'Urgence—Indications et Résultats. By J. MIALARET, Chirurgien des Hôpitaux de Paris, and G. EDELMANN, Chef de Clinique chirurgicale à la Faculté. $5\frac{1}{2} \times 9$ in. Pp. 136. 1949. Paris: Masson et Cie. 320 fr.

Clinical Radiation Therapy. By IRA I. KAPLAN, M.D., F.A.C.R., Clinical Professor of Radiology, New York University. Second edition. $6\frac{1}{8} \times 9\frac{1}{4}$ in. Pp. 844 + xvi, with 393 illustrations. 1949. New York: Paul B. Hoeber Inc. $15.00.

Die individuelle Reaktionsweise bei chirurgischen Infektionsprozessen. By Dr. GERD HEGEMANN. $6\frac{1}{2} \times 9\frac{1}{2}$ in. Pp. 126 + iv. 1949. Berlin: Springer. DM 12.

Preoperative and Postoperative Care of Surgical Patients. By HUGH C. ILGENFRITZ, A.B., M.D., F.A.C.S., Formerly Assistant Professor of Surgery, Louisiana State University School of Medicine, and Visiting Surgeon, Charity Hospital of Louisiana at New Orleans. With a Foreword by URBAN MAES, M.D., D.Sc., F.A.C.S. $5\frac{1}{2} \times 8\frac{3}{4}$ in. Pp. 898, with 110 illustrations. 1949. London: Henry Kimpton. 52s. 6d.

A Descriptive Atlas of Radiographs. By A. P. BERTWHISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Seventh edition. $7 \times 9\frac{3}{4}$ in. Pp. 622 + xxx, with 979 illustrations. 1949. London: Henry Kimpton. 50s.

Anévrysmes artériels et Fistules artério-veineuses. Physiologie pathologique et Traitement. By RENÉ LERICHE, Professeur au Collège de France. $6\frac{1}{2} \times 9\frac{1}{2}$ in. Pp. 312, with 50 illustrations. 1949. Paris: Masson et Cie. 900 fr.

Maladies du Squelette. By LUCIEN LEGER, R. DUCROQUET, and HENRY LEGER. $6\frac{5}{8} \times 9\frac{7}{8}$ in. Pp. 256, with 129 illustrations. 1949. Paris: Masson et Cie. 1200 fr.

Nursing Care of Neurosurgical Patients. By ROLAND M. KLEMME, M.D., F.I.C.S., F.A.C.S., Professor of Surgery, St. Louis University School of Medicine, St. Louis, Mo. 6×9 in. Pp. 117 + xiv, with 61 illustrations. 1949. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications Ltd.) 15s.

Cunningham's Manual of Practical Anatomy. Revised and edited by JAMES COUPER BRASH, M.C., M.A., M.D., F.R.C.S. (Edin.), F.R.S.E., Professor of Anatomy, University of Edinburgh. Eleventh edition. $4\frac{7}{8} \times 7\frac{1}{4}$ in. Vol. II: Thorax and Abdomen. Pp. 488 + x, with 238 illustrations. Vol. III: Head and Neck; Brain. Pp. 513 + x, with 230 illustrations (including 16 plates), many coloured. 1949. London: Oxford University Press. Each Volume 21s.

Advances in Surgery. Under the Direction of an Editorial Board (Chairman: WILLIAM DE WITT ANDRUS). 6×9 in. Volume I. Pp. 554 + xii, with numerous illustrations. 1949. New York and London: Interscience Publishers Ltd. 66s.

Funktionelle Pathologie in der Chirurgie. Chirurgie und vegetatives System. Eine Einführung in die korrelative Chirurgie. By Dr. CHRISTRAAN VAN GELDEREN, Amsterdam. Second edition, revised and enlarged. $6\frac{1}{4} \times 9\frac{1}{2}$ in. Pp. 206 + x, with 40 illustrations. 1949. Berlin: Springer. DM 18.60.

Tumours of Bone. By CHARLES F. GESCHICKTER, M.D., Professor of Pathology, Georgetown University Medical School, etc., and MURRAY M. COPELAND, M.D., Professor of Oncology, Georgetown University Medical School, etc. Third edition. $6\frac{7}{8} \times 10$ in. Pp. 810 + xviii, with 642 illustrations. 1949. Philadelphia and London: J. B. Lippincott Co. £5 5s.

Care of the Surgical Patient. Including Pathologic Physiology and Principles of Diagnosis and Treatment. By JACOB FINE, M.D., Surgeon-in-Chief, Beth Israel Hospital; Professor of Surgery at Beth Israel Hospital, Harvard Medical School. $6\frac{3}{8} \times 9\frac{5}{8}$ in. Pp. 541 + xvi, with 40 illustrations. 1949. Philadelphia and London: W. B. Saunders Co. 40s.

Army Foot Survey. An Investigation of Foot Ailments in Canadian Soldiers. By Colonel R. I. HARRIS, M.C., R.C.A.M.C., and Major T. BEATH, R.C.A.M.C. $8\frac{1}{2} \times 11$ in. Pp. 416, with 114 illustrations and many charts, etc. In typescript. 1949. Ottawa: National Research Council of Canada.

A Practice of Orthopædic Surgery. By T. P. MCMURRAY, C.B.E., M.B., M.Ch., F.R.C.S. (Edin.), Professor of Orthopædic Surgery, Liverpool University; etc. Third edition. $5\frac{3}{8} \times 8\frac{1}{2}$ in. Pp. 444 + viii, with 191 illustrations. 1949. London: Edward Arnold & Co. 30s.

Functional Localization in the Frontal Lobes and Cerebellum. With Particular Reference to the Operation of Frontal Lobotomy. By JOHN F. FULTON, O.B.E., M.D., D.Sc., LL.D. (Birm.). Being the William Withering Memorial Lectures delivered at the Birmingham Medical School, 1948. $5\frac{3}{8} \times 8\frac{1}{2}$ in. Pp. 140 + xii, with 53 illustrations. 1949. London: Oxford University Press. 15s.